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A STUDY ON KALLADAIPPU

(DISSERTATION SUBJECT)



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requirements to the Degree of*

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INTRODUCTION

Siddha system of medicine is one of the ancient systems Contemporaneous with those of the submerged lands, Egyptian, Mesopotamian, Chinese and Grecian medicines. The unique nature of the system is its continuous service to humanity for more than five thousand years in combating diseases, and in maintaining its physical, mental and mortal health.

The Siddha medicine is well founded on the basic principles of nature and its elements after a careful and thorough study of the human system.

The ancient Tamil in their quest for knowledge for longevity had developed two ways, by which man can achieve mastery over nature; the one is the yogic way and other through medicines. Yogies is known as Siddhars. Hence the system of medicine profounded by them came to be known as siddha system of medicine.

Siddha is a Tamil word that is derived from its root Siddhi – which means perfection in life or heavenly bliss. Siddhars attained eight kinds of supernatural powers. The persons who had attained eight kinds of miraculous power in the life are known as Siddhars.

The earliest medical treatise in Tamil was propounded by Sivanar, who was the first to preside over the ancient first Tamil academy. It was followed by a number of works of immortal Siddhars.

Siddha system dealing not only science of life, nature of universe, astronomical data, cosmic dance, atomic theory, space travel, alchemy and kayakalpa and medicines et.

Agastya siddhar who is the chief of the Siddhars' school is said to have been a celebrated philosopher and physician who laboured amongst the Tamil in southern India.

The man is said to be the microcosm and the world the macrocosm; because what exists in the world exist in man; or in other words there is nothing in the macrosom of nature that is not contained in man. So man must be looked upon as an integral part of universal nature and not as anything separate or different from the latter. In the organism of man, these forces may act in an abnormal manner and cause diseases there by. Similarly, in the great organism of the cosmos, they may act abnormally likewise and bring about disease on earth. Seven planets exercise special power over some part of the body to cause diseases or diseases accordingly to their influences on the three humours in the system.

The human body is composed ninety six thatwas or constituent principles in nature including elements, bodily and mental organs, faculties matter etc.

The three humours are vadham, pittam and kabham. They are the three fundamental principles and essential factors in the composition and constitution of the human body. Three humours are representing respectively the air, the fire and the water of the five elements. Which form the connecting link between microcosm or man and macrocosm or world? The external air, heat and water correspond to the internal vatham, pitham and kabam.

Man is thus linked with external world and any change in the elementary condition of the external world has its corresponding changes in the human organ (humours). The normal order of vadham, pitham and kabam are in the proportion of 1: ½:¼ (4:2:1) respectively. Any changes in this proportion are some to bring about disease or death. But the maintenance of their normal proportion gives vitality of the organism and assures the prevention health and longevity of life.

The siddhars materia medica is based on human pathology. It asserts that all substances of the animal, the herbs and the mineral kingdoms contain one or more of these three humours in their composition. They should play an important role in the maintenance of these humours in the men and women. Siddhars knowledge is on minerals, metals and plants suitable combination of these things on processing and the preparation of medicine. According to the classification of the five elements of nature was of a very high order as can be learned from the lines of the ancient siddha medicine.

Super intellectual siddhars devised and utilized special technique for diagnosis a disease known as *envagai thervugal*. These eight factors play a vital role in finding out disease and deranged life elements.

Urinary calculus is known to mankind since antiquity. The earliest recorded example being bladder and kidney stones detected in Egyptian mummies dated to 4800 B C.

Kalladaippu is most common disease of the present society Due to sedentary life style and abnormal diet habits.

Clinical features of ***kalladaippu*** are lower abdomen pain, dysuria, oliguria, burning micturition, nausea, vomiting, haematuria and fullness of abdomen.

In the contemporary world, till now no internal medicine is found for the renal calculus. In spite of new approach in diagnosis and management like Extra Corporeal Shock-wave Lithotripsy (E. C. S. L) and endoscopy surgery, but it still continues to cause significant morbidity with high tendency of reoccurrence and this kind of treatment procedure not reach to low socio economic status families, who are mostly affected renal calculus.

The contribution of Yoogi in the field of regional and humoral pathology regarding the disease ***kalladaippu*** is highly commendable; Siddha

medicine is ancient duration system of medicine for centuries has provided proven treatment of disease like ***kalladaippu*** without morbidity.

We must praise, safe guard and follow the Siddha system of medicine and all so furnish it to take up with the up to date advancement.

So the author of this dissertation work has selected ***kalladaippu*** under the neer arugal noi disease, expected by siddhar Yoogi in Yoogi vaithiya sindhamani perunool -800 and clinical entity comparable to urolithiosis in modern medicine. The drug ***kalladaippu thool*** was selected in Anupoga vaithiya navaneetham.

AIM AND OBJECTIVES

The aim of this work is to select the case of ***kalladaippu*** patients and administer them with the trial drug, as per the line of treatment and analysis clinically and experimentally to prove efficacy of the drugs. Siddha literature revealed so many drugs for ***kalladaippu***; among them the author has selected ***kalladaippu thool***.

The main objective of the patient study is to create awareness about Siddha medicine and highlight the efficacy of Siddha medicine for the disease of ***kalladaippu*** among the public with basic intention in mind. The following specific objectives have been drawn.

To collect and compare varies siddha and modern medicine.

To have an idea about incidence of ***kalladaippu*** with reference of age, sex, residence (Nilam), occurrence and duration of diseases.

To have a detailed study of investigation, diagnosis and prognosis of disease.

To study through investigation by Siddha fundamental and modern investigation during and after treatment in all patients.

To study the urine analysis, hematological study and ultrasonography for all patients.

To study the biochemical analysis of trial drug.

To study pharmacological diuretic study and acute toxicity study of trial drug.

To conduct the clinical trial of ***kalladaippu thool*** on ***kalladaippu***.

கல்லடைப்பு (KALLADAIPPU)

The disease **kalladaippu** is placed under siruneer noi or moothira noi.
Which is classified in to,

1. Neerina arukkal noi (Oligurial diseases)
2. Neerina perukkal noi (Polyurial diseases)
3. Neerilivu, Venner and Thelimeer.

This has been mentioned by Therayar in his 'Theran karisal' as follows

“நீரிரு வினைக் குணத்தை நீயறிவித்துச் சொல்வாம்
நீரினைப் பெருக்கலொன்று நீரினை யருக்கலொன்று
நீரிழிவுடனே கொல்லும் நீர்கட்டு வினைகளொன்று”
-தேரன் கரிசல்

Kalladaippu is one of the Neerina arukkal noigal
Verupeyar (synonym): Achmari

இயல்பு (DEFINITION)

According to the text Siddha maruthuvam by Dr. Kuppusamy.
There is gradual or suddenly obstruction to the flow of urine, pain with
burning sensation in the urethral tract, low back pain, renal angle pain, and
sands like crystal's deposit in urine. These are characteristic features of
Kalladaippu.

“தானெற்ற மூத்திரத்தால் நறநறவென்று
 தங்கியதோர் பொடியெனும் மணல்தானப்பா
 வானென்ற சிறியதொரு கல்லா வதப்பா
 வளமாக வந்துவிழும் நோய்க்குத் தானே
 ஏனென்ற அச்மரிரோக மென்ற பேராம்
 எளிதாக கல்லுக்குள்தான் விழுகும் போது
 கோனென்ற குண்டிக்காய் மூத்திரக்குழலப்பா
 குணமான மூத்திரப்பை நீர்தாரை கேளே
 கேளடா முன்குறியில் எரிச்சல் கண்டு
 கெடியாக வேதனைகள் காட்டுமப்பா
 வாளாட சிறியதொரு கற்கள் தானே
 வளமான மூத்திரப்பை குழல்வழிப் படியாயத்
 தேளடா வரும்போது திரே கந்தன்னில்
 தெரிப்பது போல் யிருவேதனை செய்யும்பாரு
 நாளடா கற்கள் தானிறங்கி விட்டால்
 நலமானவேதனைகள் தான் தீரும்பாரே”

-அகத்தியர் குணவாகடம்

Agasthiyar says the definition of **kalladaippu** as sand like crystals deposited in urine, followed by small size of stones are excreted in urine. Stones are stagnated in kidney, ureter, urinary bladder and urethra. Pain with burning sensation start in urethral orifice followed to agonizing pain occurs during the stone moving in urethral tract from the bladder, when the stone removed pain also relieved.

Large concentration of minerals in the bladder or kidney produces calculus or gravel. It is altered with difficulty in passing in urine.

- T. S. Sambasivam Pillai.

Kalladaippu is defined as pain in and around the umbilicus, fever, dysuria and urine smelling like that of goat's urine.

- Jeevanachamirtham.

NOI VARUM VAZHICAL (AETIOLOGY)

“விழுகு சிலசேரம் விடுபட்டு நீரோடும்
ஒழுகிய வாயுவு மொது கினால் நோகாது
வழுகிய மந்தத்தால் வாயுவந்தே புகில்
கழுகி முதிர்ந்திடும் கல்லடைப்பாகுமே”

-திருமூலர் கருகடை வைத்தியம்

Stone formed by derangement of humours of vatham and pittam.

“தெளிந்ததோர் கல்லடைப்புப் பத்தி கேளாய்
சிறிதுநாட் தொடங்கியே மேகந் தன்னால்
தளிந்ததோர் சலப்பையி லுதிரந் தோய்ந்து
சந்தசத் தாகவே பருத்துக் கொள்ளு
வளிந்ததோர் வாதபித்தந் தால் வந்து
பெருங்கல்லாய் நீர்வழி யடைத்து
நளிந்ததோர் நாலுவிதக் கல்லடைப்பு
நண்பான வரலாறு நாட்டக் கேளே”
-யூகி வைத்திய சிந்தாமனி

It is worth while to mention the poem of Yoogimamunivar who is authority of Siddhars regional and humoral pathology. He has revealed about this disease since 14th century.

Yoogimamunivar says that as blood clotted in urinary bladder due to urinary tract diseases followed by swelling of urinary bladder, urinary stones are formed in urinary tract induced by humour of vatham and pittham.

“நாட்டமாய்க் கற்ப்பழித்துக் கடமை வாங்கி
நலிபண்ணிக் கூடாமல் வழக்குப் பேசி
கூட்டமாய்க் குருவுடைய உடமை தன்னைக்
கொடாமலே கைக்கொண்ட கொடுமை யோர்க்கும்
வாட்டமாய் வரம்புதம்புத் திரிந்த பேர்க்கும்
மாறுபாடா யெடுத்துப்பொருள்க டனைக்
காட்டியே கைக்கொண்டு கபடு பண்ணும்
காலாந்தர் கல்லடைப்பிற் காளாவாரே”
-யூகி வைத்திய சிந்தாமனி

In this explains that mind plays a major role in causing many diseases and connection between body and mind and soul is established i.e., the mukkutram deranged by internal factors such as sexual perversion, anger and robbery.

“கலங்கினதோர் தண்ணீர்தான் குடித்த பேர்க்கும்
கல்லெலும்பு மயிர்மண்தான் கலந்தன்னத்தில்
அலங்கினதோ ரன்னங்க ளருந்த லும்
அழுகலொடு முற்றபண்ட மருந்தலாலும்,
மலங்கினதோர் மாப்பண்ட மருந்தலாலும்
மந்தத்தில் வாயுவாம் பதார்த்தந் தன்னை
துலங்கினதோ ருசிதன்னிற் சுவைத்தலாலும்
சுருக்ககாய்க்கல்லடைப்பு வந்து தோன்றுந் தானே”
-யூகி வைத்திய சிந்தாமனி

The causes mentioned here,

- Contaminated water
- Stone crystals, bone, hair and sand mixed food.
- Decayed food
- Carbohydrate food
- Humour of vali induced food

“நீரினைத் தடுத்தல் செய்யின்
நீர்கட்டுத் துவாரம் புண்ணாம்
பாறிடுஞ் சந்து சந்தில்
பண்புறு நோவதாகும்
நேரிலங்கயருங் காம்யம்
நிச்சிய நோதல் செய்யும்
பாரினி லபான வாயு
பண்புறச் சேரு மன்றே”

-சித்த மருத்துவாங்கச் சுருக்கம்

Siddha maruthuva surukkam explained that urination is one of the 14 visceral reflexes. When one suppress this visceral reflex it lapses into manifestation of morbidity, which comprises inflammation of bladder,

anuria, arthralgia, pain in genital region and deranged of keezh nokkukaal. This leads to the formation of calculus.

The author also explain that ejaculation of semen is one of the 14 visceral reflexes when one suppress this reflex it causes fever, retention of urine which favours urinary calculi, chest pain, arthralgia, and white discharge.

“சுக்கிலந் தனையடக்கின்
சுரமுடனீர்க் கட்டாகும்
பக்கமாங் கைகால் சந்து
பாரநோய் வழியிறங்கும்
மிக்கமார் நோயுண்டாகும்
மிகுத்திடும் பிரமேகந்தான்
தக்கதோர் போதுமாகின்
தரித்திடும் வாயுக் கூறே”
-சித்தமருத்துவாங்கச் சுருக்கம்

“கரு நீரடக்கல் விரையில் அடிபடல்
நீரியந்தாக் கல் சிறு நீரடக்கல்
வளிநோய் மிருக்கு முணவும் ஒழுக்கமும்
கடைப் பிடித்திடுதல் மேகமுதற் பல
பிணியுறல் எழுமிவை யடிப்படையாகக்
கல்லடைப் பொன்னுங் கடும்பிணி விளையும்
வளியது மீறியை யொடு மல்லாது
கருந் ரொடுங் கலந்துற நீரகத்துச்
சிறுநீர்க் கழிவு தொகுத்த லாலும்
அன்னவை கல்லெனத் திரளு மென்ப”
-நோய் விளக்கம்

According to noi vilakkam

- Derangement if humour in blood,
- Excessive indulgence in sexual activity or sexual perversion,
- Trauma on testes,
- Suppression of urine and semen

- Inflammation of bladder
- Syphilis (Mega noi)
- Stagnation of urine in urinary tract.
- Dryness of semen causes the formation of stones.
- Increased intake of food that cause flatulence.

POTHU KURI KUNANGAL

- Gradual or sudden obstruction to flow of urine,
- Unbearable pain(agonizing pain) in the penis
- Excruciating pain and swelling is experienced at tip of penis if the calculus attempts to expel.
- Colicky pain radiating from loin to groin, lower abdomen, urethra and genitalia if the calculus is irregular with sharp projection.
- Burning and scanty micturation and haematuria.

SYNDROMES ASSOCIATED WITH KALLADAIPPU

உக்கார சூலை

“குத்துமுகக் காரசூலையின் குணந்தான்
கோர்வையாய் விலாவதனில் முதுகில் நெஞ்சில்
அத்தியினில் நாபியில பானமாங் குதத்தில்
அதிகத்துன் மாங்கிசந்தான் வளர்ந்து மேவிப்
பத்துமணற் படுக்கைபோற் சலத்து வாரப்
பதிநெருக்கி மூத்திரமாங் கிரிச்சி யுண்டாய்த்
தத்துசடங் கடுப்பெடுத்து மதிக லங்கித்
தளர்ச்சியொடு மயக்கமாய்த் தள்ளுந்தானே”

Excessive growth of muscles in chest region, back of trunk, umbilicus and anal and urethral orifice followed by stricture of urethral orifice like a sand like crystals blocked in urethra. Dysuria, body pain, impairment of conscious, tiredness, and giddiness occurs.

CLASSIFICATION OF KALLADAIPPU

“தோன்றினதோர் நாலினிட நாமங் கேளாய்
விருக்கான வாதத்தின் கல்லடைப்பு
பூன்றியதோர் பித்தத்தின் கல்லடைப்பு
புரண்டதோர் சேட்டுமத்தின் கல்லடைப்பு
தீன்றியதோர் தொந்தமாங் கல்லடைப்பு
தேகத்தை பற்றியே சிறிது காமம்
தான்றியே சலப்பை யில்வந்தி ழிந்து
சருவியே லிங்கத்திற் றரிக்குந் தானே”

The most experienced of Siddhars, Yoogimamunivar who has studied the disease according to regional and humoral pathology classifies **kalladaippu** in to 4types there are

1. வாத கல்லடைப்பு,
2. பித்த கல்லடைப்பு,
3. ஐயக் கல்லடைப்பு,
4. முக்குற்ற கல்லடைப்பு.

1. வாத கல்லடைப்பு (VALI KALLADAIPPU)

“தரித்துநா பிக்குங்கீழ்ச் சுருக்காய்க் குத்திச்
சலமலந்தான் வீழாமற் றம்பமாபகி
வரித்துமே லிங்கத்தில் வலியுமாகி
மருவியதோர் பொத்தியெலாஞ் சுரந்து கட்டி
திரித்தியே கிடகொடாப் புரட்ட லாகித்
தேம்பியே மூச்சுமாய் வயிறு முப்பும்
உரித்ததோர் சதைபோல உவர்ப்பு மாகும்
ஓங்கியதோர் வாதக்கல் லடைப்பு தானே”

- Retention of urine.
- Lower abdomen pain.
- Pain with swelling of penis.
- Agonizing pain.
- Breathlessness.
- Abdomen distention.
- Excretion of urine with mucus.

2. பித்த கல்லடைப்பு (AZHAL KALLADAIPPU)

“அடைப்பாகிச் சலந்ததானு மருவலாகி
அயங்காய்ச்சிச் சொருகினாற் போலே காணும்
புடைப்பாகப் பொத்தியெங்கும் புழுக்க மாகிப்
பூட்டுப்போல் பிசுவாகிப் புரட்ட லாகும்
மடைப்பாகி உதிர நிற மாய்க்கல் லாகி
வந்திழிந்து லிங்கத்தில் மாட்டிக் கொள்ளும்
குடைப்பாகிக் குற்றலாய்க் கூச்ச லாகிக்
குதட்டுமே பித்தக்கல் லடைப்புத் தானே”

- Stone blocked in urinary tract followed by oliguria.
- Pain in urethra like an insertion of burning iron bar, followed by sweating all over body.
- Anuria, Agonizing pain.
- Blood stained calculus stagnated in urethra followed by piercing and boring pain with sensitiveness.

3. ஐயக் கல்லடைப்பு (IYYA KALLADAIPPU)

“தானான தொப்புளிலே வில்லு போலச்
சலியாமற் சுரந்துமே சற்றே குத்தும்
ஏனான காலோடு கைகள்சந்து
இடுப்புதான் குடைச்சலா யிசிவு காணும்
வேனான லிங்கத்தின் வேண்மை தன்னில்
விறுவிநென்றே கடுப்பாகி வியர்வை யாகும்
தேனான வெளுப்புக்கல் சிறுகல்லாகச்
சிக்கலாய் வந்திறங்குஞ் சிலேட்மந் தானே”

- Abdomen distention like a bow with pain.
- Pain in all joints of upper and lower limb and hip joints.
- Pain in penis, sweating all over body.
- White coloured stone excreted in urine.

4. முக்குற்ற கல்லடைப்பு (MUKKUTTRA KALLADAIPPU)

“வந்திறங்கும் நீர்த்தாரை யடியிற் றானும்
மாவருத்த முண்டாகி வலியுமாகி
நொந்திறங்கி நீர்தானு மருவிப் பாயும்
நொய்தான சிறுமணல்போல் நொறுங்கிக் கல்லாஞ்
சந்திறங்கி நீர்வழியில் வந்து வீழும்
தாக்கான சிறங்கைக்கல் தினமொன்றுக்கு
துந்திறங்கித் தினந்தினமு மிழிந்து கொல்லும்
தொந்தமாங் கல்லடைப்புச் சூட்டிடாயே”
(யூகி முனி)

- Severe pain in urethra.
- Dysuria.
- Oliguria.
- Crystals excreted in urine like a small sands.
- Handful crystals excreted in urine daily.
- It is fatal disease.

CLASSIFICATION IN DHANVANTHIRI VAITHIAM

“திருந்திய வாதபித்தச் சிலேற்பனம் பிரகோபத்தால்
வருந்த சுமரித்தா னான்கு வகைபடுங்கல்லெரிப்பான்
பிரிந்திடுஞ் சிலேற்பனா சுமரி பித்தாபின்னு
மிருந்திடு சுக்கிலாசுமரி நான்கு மெய்துமென்றே”
-தன்வந்திரி வைத்தியகாவியம்

In Thanvanthiri vaithiyam, **kalladaippu** is classified to four types they are

கல்லெரிப்பான் (KALLERIPPAN)

“கத்துநீர் நாளந்தன்னிற் சுக்கிலந்தனிற் சிலேற்பம்
பித்தமீ துவர்த்தல் கல்லாய்ப் பிசுகினீரடைத்து கொள்ளுங்
கொத்துநீ ரிற்றுவிழுங் கொப்புள்ளோ குடம்புகாயுந்
சித்தமா யருசியுண்டாந் சேர்ந்த கல்லெரிபாமே”

- Increased iyyam and azhal kutram dries the urine and semen forming calculi
- Sudden or gradual obstruction in urinary tract
- Dysuria
- Pain in umbilicus
- Fever
- Anorexia.

சிலேத்தும அச்மரி (SILETHUMA ACHMARI)

“நீர்வரு நாளந்தன்னில் நின்றநீர் சிருத்துகொண்டு
சோர்தரும் சிலப்பு வெண்மை சுக்கிலம்போல்வீழும்
பேர்பெற நாலா மெட்டுப் பின்னமாய்க் கல்லு வீழும்
எர்பெறு சிலேற்பனத்தின் அசுமரி என்னலாமே”

- Calculus in the Ureter or urethra causes hydronephrosis
- Oliguria,
- Reddish white in colour and falls out like semen
- Stones are expelled as 4 or 8 fragments.

பித்த அச்மரி (PITHA ACHMARI)

“செய்யும்நீர் நாளந்தன்னில் பித்தத்தா லெரிப்பெழுந்து
செய்யுவுஷ் ணத்தால் வெந்து சேங்கொட்டைபோல் கல்லுன்டாம்
நய்யவே தரனகள் செய்யும் நவில்குணம் பித்தந்தன்னில்
எய்த்சுமரி யென்றேமுன் நியம்பின ரறிவின்மிக்கோர் “

- Burning sensation in urethra due to azhal kutram
- Burning micturition
- Formation of stone that appear like semicarpus anacardium seeds.

சுக்கில அச்மரி (SUKKILA ACHMARI)

“சுக்கிலம் வருங்காலத்தில் தம்பித்தாற் சிக்கிலந்தான்
மிக்கக்கல் லாகி வெதும்பி விதனமாய் நீர்விடாமற்
சிக்கி நீர் விழாமலங்கே மணல்விழும் வெளுக்கும்தேகம்
மிக்குண ஞ்சுக்கிலாசு மரிய சாத்திய மென்றோதோ”

- Suppressions of semen during ejaculation, develops on to stones and obstruction in the flow of urine
- Sand like gravels are expelled
- Pallor of the body.
- This is incurable.

CLASSIFICATION ACCORDING TO NOI VILAKAM

“வளி முதல் மூன்றினுந் தோன்றலாலும்
கருநீர் தன்னீர் தோன்றலாலும்
கல்லடை நால் வகைப் படுமெனமொழியே”

There are four types of **kalladaippu** according to noi vilakkam

1. Vali kalladaippu,
2. Analakalladaippu,
3. Iyya kalladaippu,
4. Karuneer kalladaippu

வளிக்கல்லடைப்பு (VALI KALLADAIPPU)

“படர்மிகப்படுத்தல் பற்கள்கடித்தல்
நடுங்கல் உந்தியும் குறியும் பிசைதல்
கசடுகீழ் சளியொடு கழலல் அமுதல்
சிறுநீர் துளித்தல் என்பவும் பிறவும்
வளியின் கல்லடைக் குறியேன மொழிய
கறுத்துந் சிவந்தும் முனைகள் பரந்தும்
வளியின் கல்லது வடிவுனு மென்ப”

- Tongue biting , palpitation and shivering,
- Crushing of the lower abdomen and genital organs
- Dribbling of urine.
- The stones are blackish red colour.

அனலக்கல்லடைப்பு (ANALA KALLADAIPPU)

“சுட்டென நீரியம் மிகவெம்பிடுதலும்
நோதலும் அவைக் கல்லடைக்குறியே
சிவந்துங் கறுத்து மஞ்சளாகியும்
சேங்குரு வடிவில் கல்லது தோன்றும்”

- Burning micturition,
- Dysuria,
- The stones are reddish black or yellow in colour and passing of smalls stones

ஐயக்கல்லடைப்பு (IYYA KALLADAIPPU)

“நீரியங் குத்தல் திணித்தல், குளிர்த்தல்
எனுமிவை ஐயக் கல்லடைக் குறியே
வெளுத்தும் தேனிறமாகிய மொளிர்ந்தும்
பெரு வடிவுடைத்தாம் ஐய கல்லடைப்பு”

- Pricking pain, forceful pain with severe intensity when passing urine,
- Fever with rigors,
- White or honey coloured shining or luminant large size stone expelled.

கருநீர் கல்லடைப்பு (KARUNEER KALLADAIPPU)

“கரு நீர்க்கல்லின் வளி சினந்தெழுந்து
விரைகளி னடுவில் அதுதனைத் தடுத்தலின்
கருநீர்க் கல்லடை மருவிடு மென்ப
நீரியம் நோதல் சிறுநீர் தடைபடல்
விரை வீங்கிருத்தல் எலுமிவை பிளவும்
கருநீர் கல்லடைக் குறியென மொழிய
கருநீர்க் கல்லினை வளியது முடுகிச்
சிறியவும் பெரியவுந் துண்டுகளாக நொறுக்கிடும்
அவை சிறுநீர் வழி வெளிப்படவாகும்
அவை சிறுநீரினைத் தடுத்தல் நிற்கும்
சாற்றிய நீரினை தடுத்து நிற்பின்
ஆற்றல் குறைதல் வயிறு நோதல்

சுவைகெடல் வெளிறு மறுப்பு நீர்வேட்கை
வெல்வளி யெனுமிவை விளைந்திடு மென்ப
சிறுவர்க் காயின் கல் சிறிதாதலின்
கருவியி னெடுத்தல் எளிதா மென்ப
கருநீர்க் கல்லடை சிறுவர்க் கில்லை
விரையும் உந்தியும் மிகைப்பட வீங்கல்
சிறுநீர் தடைப்படல் நோவு மிகுதல்
என்பவை துணிகல் மணலிடைத் தோன்றின்
பிழைத்த வரிதெனப் பேசுவர் புலவர்”

- Sudden or gradual obstruction to flow of urine,
- Swelling of testis,
- Excessive vali kutram breaks the stones into small and large size crystals and expels along with urine.
- Sudden stoppage of urine stream,
- Retention of urine,
- Abdominal pain,
- Loss of taste excessive thirst,
- Pricking pain with swelling of abdomen and testis,
- Retention of urine or anuria may leads to renal failure and fatal

Classification in Jeevaratchamirtham and Anubhava vaithiya devaragasium:

Fives types

1. Vatha achmari,
2. Pitha achmari,
3. Kabha achmari,
4. Shukila achmari,
5. Swargara achmari.

Classification of disease in Indian medicine

-By Dr. Juliusjully

Four types;

1. Vatha achmari,
2. Pitha achmari,
3. Kabha achmari,
4. Shukila achmari.

Classification in siddhar aruvai maruthuvam- four types:

1. Vali kalladaippu,
2. Azhal kalladaippu,
3. Iyya kalladaippu,
4. Venneer kalladaippu.

MUKKUTRA VERUPADUKAL (PATHOLOGY)

The imbalance in one's diet and fluid intake increases the Azhalkutram. This raised kutram dries up the body fluid and urine resulting in concentration of salts; this further affects the keezh nokku kaal. One of the functions of the keezh nokku kaal is to excrete urine. So when this keezh nokkukaal is affected urine will be obstructed within urinary tract. This favors the deposition of urinary salts to develop in to calculi any where in the kidney or urinary tract.

நோய் கணிப்பு முறை (DIAGNOSIS AND PROGNOSIS)

In piniyarium muraikal the following principles are followed in Siddha system. There are

- 1.பொறியாற்றேர்தல்,
- 2.புலனாலறிதல்,
3. வினாதல்.

The maruthuvar (physician) should observe the patient, palpate and interrogate the patient thoroughly. This is stressed also understood by this maxim.

“Eyes first and most, Hands next and little, mouth last and never”

I, II, PORIYAL ARITHAL AND PULANAL ARITHAL

Poriyal arithal or understanding by the five organs of perception.

Pulanal arithal or understanding by the sense objects. There are

1. Mei- Ooru (Somatic sense)
2. Vaai – Suvai (Taste)
3. kan – Oli (Vision)
4. Mookku – Natram (Smell)
5. Sevi – Oosai (Sound)

1. வினாதல் (INTEROGATION)

An effective history taking helps one to diagnosis properly. By vinathal the physician should ask the patients native place, mode of living, food habits, personal habits, complaints and duration of illness etc. If the patient is deaf or dumb or if the patient is a child, the particulars should be obtained from his relatives or parents.

Poriyal arithal, pulanal arithal and vinathal are applied through eight special tools of investigation that is envagai thervugal.

ENVAGAI THERVUGAL

“நாடிப்பரிசம் நாநிறம் மொழிவிழி
மலம் மூத்திரமிவை மருத்துவராயுதம்”
-அகத்தியர் நாடி

NAADI

The word pulses means the beating of artery felt with the tip of the fingers, its rate and character go to indicate a person's condition of health. It is also understood as the beating, throbbing or rhythmical dilatation of arteries as the blood is propelled along them by the contraction of the heart in the living body. The term pulse in medical practice is usually applied to beat or throb felt in radial artery at the wrist; through it may felt over the temporal, carotid, ulnar, brachial, femoral and other artery.

Normally the pulse is recorded in the radial artery in the right hand for the male and left hand for the female by keeping the index finger, the middle finger and the ring finger on it after gently scrubbing the area. It is one unit in vali as felt by index finger and a half unit in azhal as felt by the middle finger and one fourth units in iyya as felt by the ring finger. The different diseases could easily be diagnosed with aid of the pulse.

“நாடி என்றால் நாடியல்ல நரம்பில் தானே
நலமாகத் துடிக்கின்ற துடிதானு மல்ல
நாடி என்றால் லாதபித்த சிலேற்பனமுமல்ல
நாடி எழுபத் தீராயிரந் தானுமல்ல
நாடியென்றால் அண்டரெண்டமெல்லாம்
நாடி எழுவகைத் தோற்றத்துள்ளாய் நின்ற
நாடியது யாராய்ந்து பார்த்தாரானால்
நாடியுறும் பொருள்தெரிந்து நாடுவாரே”
-அகத்தியர் நாடி

கல்லடைப்பு நாடி

Aggravation of valinadi produces symptoms of *kalladaippu*. This is emphasized in Agathiar nadi, Sathaga nadi and Rathina churukkanadi.

“அறைந்தோம் வாதரோகியுடல்
அடிக்கண் முகமும் பலமலமும்
நிறைந்த விழியில் நீர்வடியும்
நீண்ட நாவு கறுத்திடவும்
நிறைந்த முள்ளாய் தானிருக்குந்
சிறுநீர் பொருமி கருத்து வரும்
உறைந்த நீருங்கரு கருத்து
முறையாய் ரோகமு முண்டாய்”
-அகத்தியர் நாடி

“வாதமெனும் நாடியது தோன்றில்
சீதமந்தமொடு வயிறு பொருமல் திரட்சிவாயு
சீதமுருங் கிராணி மகோதரம் நீராமை
திரள்வாயு சூலை வலிகடுப்புத்தீரை
நீதமுருங் கிருமிகுன்மம் அண்ட வாதம்
நிலையும்நீர்க் கிரிச்சரங்கள் தந்து மேகம்”
-சதக நாடி

“மேவிய வாதஞ்செய்யும் குணத்தை விரும்பிக்கேளு
தாவிய வயிரு மந்தந் சந்துக்கால் பொருத்துநோவாம்
சேவிய தாதுநாசச் சிறுத்துடன் சிறுநீர் வீழும்
காவியங் கண்ணீனாளே மலமது கருக்கிகானும்”
-இரத்தின சுருக்கநாடி

Aggravation of azhal nadi produces symptoms of kalladaippu,

“ஏவலாய் குழலாய் பித்த செய்குணம் விளம்பக்கேளாய்
கோலவேல் விழி சிவந்து குளிந்திருக்கு மல்லால்
சீலவே நீர் கருத்து நொந்து சுறுக்கென வந்துவிழும்
ஞாலமே கிறுகிறென்று நாவுலர்ந்திருக்குந்தானே”
-இரத்தின சுருக்கநாடி

“பித்தரோகி பெருமுடல் சூடாகும்
நித்தமா முகம்நேர் விழி நாவுபல்
முத்த நீரு முயர்ந்த சிவப்பாகும்
சுத்த மஞ்சளாய்த் தோன்றிடக் கண்டிடே”

Derangement of vali-azhal naadi produces symptoms of kalladaippu.

“பொருளான வாதத்தில் பித்தஞ் சேர்ந்து
பொருந்து குணங்களா முஷ்ணவாயு சத்தி
செரியாமை புளித்தேப்பம் பொருமல் நீரிற்
சிவப்புமலம் பிடித்தலுருதாது நடட்டம்”
-சதக நாடி

ஸ்பரிசம்

By sparism the temperature of skin (thatpam-cold or veppam-heat), smoothness, roughness, sweating, dryness, hard patches, swelling, abnormal growth of organs and tenderness can be felt.

In **Kalladaippu** patients tenderness over the lower abdomen, renal angle and lumbar region, swelling can be felt in valikalladaippu (may be due to hydronephrosis)

Also patient's temperature is increased in lower abdomen and in ayyakalladaippu, sweating all over the body at the time of colic.

நா (Tongue)

By the examination of the tongue its colour, size, shape, coating, moisture, movement, ulcer, fissures, crust and condition of teeth and gums can be examined. In **kalladaippu** if there is constipation (valikalladaippu), the tongue would seem to be coated. Loss of taste in karuneer kalladaippu.

“கரு நீர்க்கல்லின் வளி சினந்தெழுந்து
சுவைகெடல் வெளிறு மறுப்பு நீர்வேட்கை”

நிறம் (Colour)

Colour of the skin all over body, a local region of affection in conjunctiva, tongue, nail bed and hair etc.

Vali udal- Block colour,

Azhal udal- Yellow or red colour,

Iyya udal – White or yellow

In kalladaippu niram of udal depends up on the body constitution, pallor of the body is observed in sukkila ashmari.

“சிக்கி நீர் விழாமலங்கே மணல்விழும் வெளுக்கும் தேகம்
மிக்குண ஞ்சுக்கிலாசு மரிய சாத்திய மென்றோதோ’

மொழி (Speech)

By examining mozhi (speech), characters, hoarseness, slurring speech, various disorders of speech such as dysarthria can be noted. In **kalladaippu** there is low pitch voice due to agonizing pain in lower abdomen and burning sensation.

விழி (Eye)

Examine the colors of eye - like reddish or yellowish discoloration and characters like dryness and lacrimation. Tiredness and redness due to pain is observed in patients with renal colic. In addition one has to be

examine the patients acquires, there may be pallor of eyes due to gross haematuria.

மலம் (Stools)

By examining malam, its nature, colour, quantity and presence of blood or pus can be noted. In valikalladaippu constipation and oliguria will be present.

“தரித்துநா பிக்குங்கீழ்ச் சுருக்காய்க் குத்திச்
சலமலந்தான் வீழாமற் றம்பமாபகி”

நீர்குறி (Urine examination)

Urinary examination is good diagnosis method compare to naadi and other Envagai thervugal. Theraiyar mention below as

நீர்குறிச்சிறப்பு
“தர்க்கசாத் திரிக ளானோர்
தங்களிற் றேர்ந்து நாடி
வர்க்கமாம் நாடி தன்னில்
வருவது மயக்க மென்றே
உற்றநீர்ப் பரிசை யாய்ந்தே
யுரைத்தன ரிதற்கு நேராய்
மற்றொரு விதிநா லில்லை
மருத்துவக் கலைவல்லோர்க்கே”
-சித்த மருத்துவாங்கச் சுருக்கம்

Siruneer should be collected in early morning; patient should be eating six tastes of food with regular time and well sleeping over night, urine should be examine with in 3¼hrs. This is quoted as

“அருந்துமாரிதமும் அவிரோதமதாய்
அக்கல் அலர்தல் அகாலவூன் தவிர்ந்தழற்
குற்றளவருந்தி உறங்கி வைகறை
ஆடிக்கலசத் தாவியெகாது பெய்
தொருமுகுர்த்தக் கலைக்குட்படு நீரின்
நிறக்குறி நெய்குறி நிருபித்தல் கடனே”

-சித்த மருத்துவாங்கச் சுருக்கம்

Siruneerin pothugunam:

“வந்த நீர்க்கரி எடை மணம் நுரை எஞ்சலென
றைந்திலுளவவை யறைகுது முறையே”
-சித்த மருத்துவாங்கச் சுருக்கம்

1. Niram (Color), 2. Eadai (Specific gravity), 3. Nurai (Froth), 4. Natram (Smell), 5. Enjal (Deposits).

Above the five parameters by which each urine sample should be examined.

NIRAM (COLOUR)

NIRA THOGAI

“பீதம் செம்மைபைங் கருமை வெண்மையென்
றோதைங் கொழுமையை யொத்துகு நீரே”
-சித்த மருத்துவாங்கச் சுருக்கம்

1. Yellow,
2. Red,
3. Green,
4. Black,
5. White.

Urine may be any colour mention above.

கல்லடைப்பு நீரின் குணம் (COLOUR INDICATING URINARY STONES)

The urine colour would look like flesh washing water this is indicated in kidney diseases.

“தீப்புலால் கழுநீர்ச் செயலெனிர் குண்டிக்
காய்த்துர்ப் பலத்தால் கதித்த நீராமத்
துர்ப்பலக் கபமும் சோரியும் கொதிப்புறப்
பற்பகலாகப் பையப் பதிந்ததே
-சித்த மருத்துவாங்கச் சுருக்கம்

EADAI (SPECIFIC GRAVITY)

Urine not thick is considered healthy.

“மிகத் தடிப்பும் மிகத் தேறலும் இன்றெனில்
சுகத்தைத் தரும் மெய்ச் சுபாவ நீர் நன்றெ”
-சித்த மருத்துவாங்கச் சுருக்கம்

NURAI (FROTH)

“பந்தமெய்ப் பசையிளகப்படும் பருவத்
தந்தர்ப் பூதமாய் அனில மூத்திரத்தில்
சம்பந்தப்படும் ததிநுரைப் புனலே”

Urine may be frothy in nature, if it is reduced in vali, azhal and ayyam are said to be deranged.

NATRAM (SMELL)

மணவிலக்கணம்

“ஓதமணத்தோ டவவோத மொத்தி றங்கும்
சீதளங் கம்மிய தேகிகளுக்கே
காணிதில் சீமுற் கலந்திழி மணமுறின்
கருப்பநா பிகளுளுங் காமநா ளத்துளும்
விரணமுண் டின்றேல் எய்து மசுமரியல்
திருத்தலே திண்ண மெனமனத் துன்னே”

-சித்த மருத்துவாங்கச் சுருக்கம்

Foul odour with pyuria is observed in patients with urinary lithiasis associated with urinary tract infection and ulcer.

ENJAL (DEPOSITS)

If urine excretion look like curd water white color and sand like deposits in urine indicate stones in kidney. This mention as

“நார்த்ததி நீர்பால் போல
நவையுற்றந் கிழியு மானால்
மாரற்ப முற்ற நீரி
லடி மண்டிக் கிடந்த தானால்
பாரிந்த மெழுகு மாங்காய்
பற்றிய கல்லி னாலே
சீருற்ற செய்கை யென்று
தெரிவுறச் செப்ப லாமே”
-சித்த மருத்துவாங்கச் சுருக்கம்

NEI KURI

The urine kept on the kidney tray in sun light, on non wind condition, should be examined by dropping a drop of gingili oil gently with rod. If oil spread like snake it indicates valineer, a ring indicates azhal neer, and a float like a pearl it indicates iyya neer and sinks in urine indicates mukkutram.

“அரவென நீண்டினக்தே வாதம்”
“ஆழி போற் பரவின் அக்தே பித்தம்”
“முத்தொத்து நிற்கின் மொழிவதென்கபமே”
-சித்த மருத்துவாங்கச் சுருக்கம்

In **kalladaippu** patients, oil spreading like ring indicates Azhal neer or snake indicates Vali neer.

சாத்தியம், அசாத்தியம் (PROGNOSIS)

“சிட்டியசாத் தியத்தைச் சொல்லக் கேளாய்
சுளுக்காகும் வாதத்தின் கல்லடைப்பு
பூட்டிட்ட பித்தத்தின் கல்லடைப்பு
புகழானசேட்டுமத்தி கல்லடைப்பு
முட்டிட்ட இதுமூன்றும் சாத்திமாகி
முனையான மருந்துகளிற் செம்மை யாகும்
தோட்டிட்ட தொந்தமாங் கல்ல டைப்புத்
தொடுசுறவே கொல்லுமிது சூட்சந் தானே”

According to Yoogimamunivar, Vali, Azhal, and Ayya kalladaippu are curable where as Mukkuttra kalladaippu is in curable.

மருத்துவம் (LINE OF TREATMENT)

“வைத்தியச் செயல் வைத்தியமே”

- திருமூலர் 800

The main object of treatment is to bring down the deranged mukkutrams to natural equilibrium by giving pugnatives, which cure derangement of vatham, this one of the cause for **kalladaippu**.

“பேதியால் வாதம் தாமும்

வாந்தியால் பித்தம் தாமும்

அஞ்சனத்தால் கபம் தாமும்”

- மருத்துவம் -II

As per the above mention, author gives purgation to all patients as their body condition. The author of dissertation has selected trial drug **Kalladaippu thool**, dose of 5gms two times per day with raddish juice for 2 months period.

In siddha system treatment is not only for removable of disease but the prevention and improving the body condition after removal of disease. This is said as kappu, neekkam and thiraippu.

DIET

All patients are strictly advised to follow this diet restriction as highly calcium and oxalate diet likes cabbage, cauliflower, tomato, pees, cashionuts, almonds, grapes, strawberry, pine apple, tea and coffee. Calcium and protein diet likes milk, milk products and non vegetarian foods.

URINARY CALCULUS

PHYSIOLOGICAL ANATOMY OF THE KIDNEY

The kidneys are two bean shaped organs situated retroperitoneal, one on the either side on the posterior abdominal wall. Each kidney weighs about 120 to 150gms and is enclosed in thin but tough fibrous capsules, and concave medial side of the kidney is the hilum where the renal artery enters and renal vein and ureter leave the kidney. The components of kidney are arranged in three layers.

1. Outer cortex

This is dark and granular appearance. It contain renal capsules and convoluted tubules, at intervals cortical tissues penetrate medulla in this form of columns is called column of Bertini.

2. Inner medulla

This gives radially striated appearance as it contains tubular and vascular structures. Medulla mass is divided in to 8 to 18 medullary or malpighian pyramids, the base of pyramids and are connect with cortex and apex projects in to minor calyx.

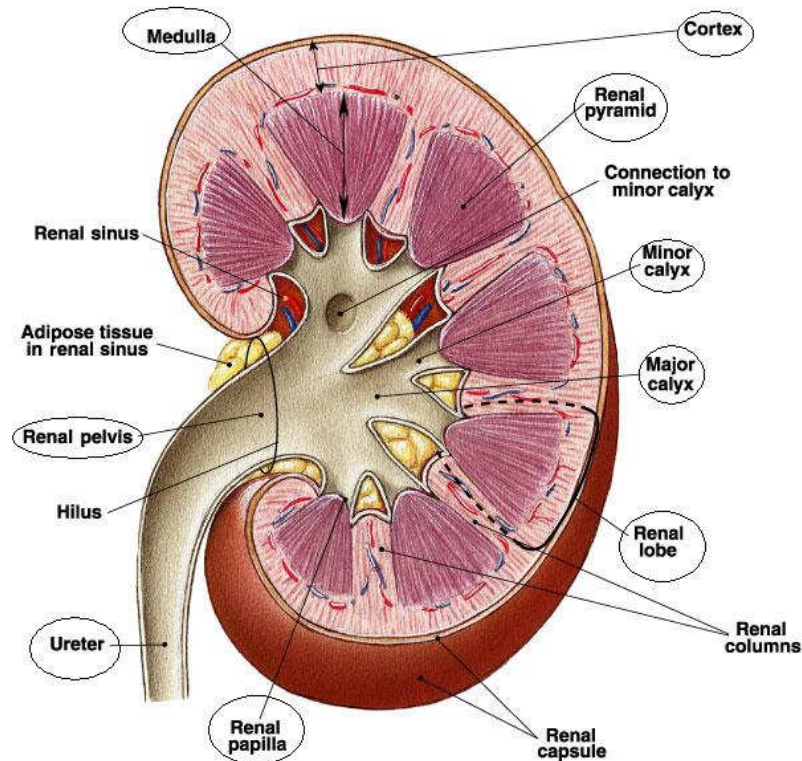
3. Renal sinus

It consist upper expanded part of ureter called renal pelvis, it subdivided into about 8 minor calyces, and branches of nerves, arteries, and tributary vein and loose connective tissues and fat.

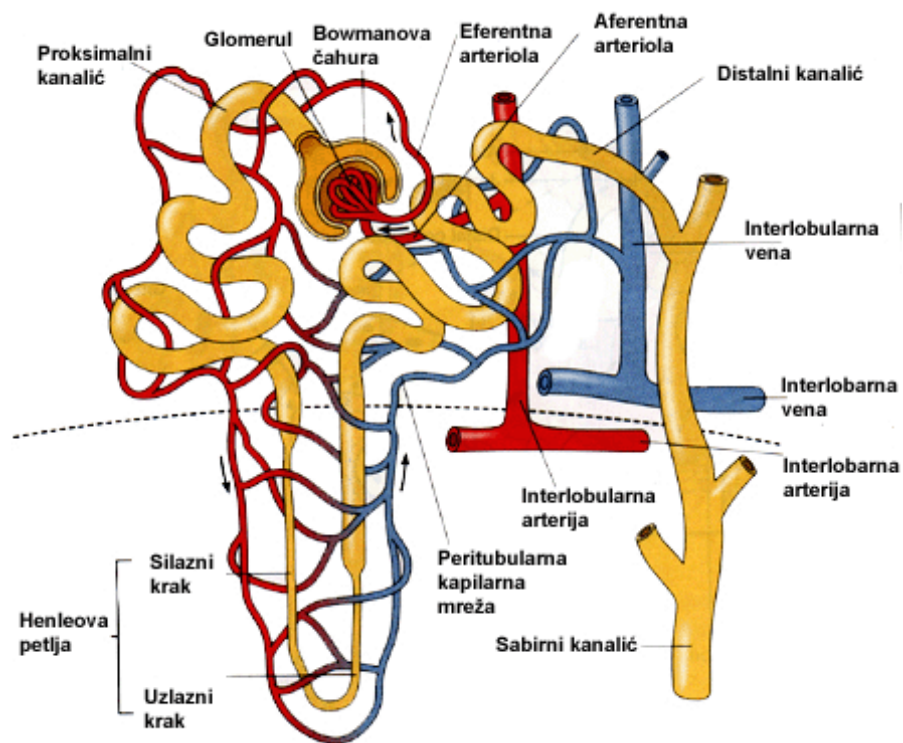
Nephron

Structural and functional unit of the kidney is nephron. Each kidney has a million nephrons. Each nephron begin in cortex as

- A funnel like dilatation called the Bowman's capsule, which enclose a tuft of capillaries the glomerulus. The Bowman's capsule together with the glomerulus is called the Malpighian corpuscle or Renal corpuscle.
- The renal tubule leaves the Bowman's capsules and becomes convoluted to form of the proximal convoluted tubule (PCT).



- It then becomes straight and passes down in the medulla as the descending limb of the loop of the Henle, after varying distances before reaching the end of the papilla, it turns round in the form of a U-shaped bend, forming loop of Henle, and passes upward towards the cortex, parallel with its former course as the ascending limb of the loop of Henle. Each limb has an outer thick and inner thin portion.
- Thick ascending limb approaches its own glomerulus, and contact with the afferent and efferent arterioles to form the juxtaglomerular apparatus. It then becomes convoluted to form the distal convoluted tubule.
- The DCT then straightens out and joins by short connecting ducts to form the collecting tubules or collecting ducts (CD). They unite to form larger collecting ducts and descend parallel to loop of Henle and open in the papilla or the duct of Bellini in the renal pelvis. The length of the nephron varies from 4 to 6.5 cm.



RENAL CIRCULATION

Renal artery arises from the aorta, enters the kidney at the hilum and divided into an anterior and a posterior branch, which give rise to about five segmental arteries. The segmental artery divided into interlobular arteries, which pass outward in the medulla between the pyramids to reach boundary zone between the medulla and cortex. Here they turn to take a horizontal course uniting with adjacent arteries to form arterial arches called arcuate arteries. Several straight arteries arise from these arches and run radially outward through the cortex. These are called interlobular arteries. From each interlobular artery, numerous afferent arteries arise, and enter the Bowman's capsule forming glomerular capillary tuft. The afferent arterioles divide into 4 to 5 large capillaries. Each large capillary divided into small capillaries, which form the loop, and capillary loop unit to form the efferent arteriole, which leaves the Bowman's capsule.

The efferent arterioles give rise to renal portal system. The efferent arterioles form a second capillary net work surrounding the tubular portion of the nephron; the capillaries of second set are called peritubular capillaries.

The tubular portion of juxtamedullary nephron is supplied by some specialized peritubular capillaries called Vasa recta. Vasa recta arise directly from the efferent arteriole of the juxtamedullary nephrons. The peritubular capillaries drain in to the venous system, which include the peritubular venules, interlobular veins, segmental vein and finally the renal vein

URINE FORMATION

Kidney excreted the unwanted substances including metabolic end products and those substances, which are present in excessive quantities in body, through urine. Normally about 1 to 1.5 liters of urine is formed every day. The mechanism of urine formation includes various processes. First, when blood passes through glomerular capillaries, the plasma filtered in Bowman capsule, when this filtrate passes through the tubular portion of nephron, it undergoes various changes both in quality and in quantity, many wanted substances like glucose, amino acid, water and electrolytes are reabsorbed from the tubules, this process is called tubular reabsorption. And some unwanted substances are secreted in to the tubule from peritubular blood vessels. This process is called tubular secretion. The urine formation includes three processes.

1. Glomerular filtration
2. Tubular reabsorption
3. Tubular secretion

1. GLOMERULAR FILTRATION

When blood passes through the glomerular capillaries the plasma filtered in to the Bowman's capsule. All the substances of plasma are filtered except plasma proteins. The filtered fluid is called Glomerular filtrate. During filtration the substances pass through a three layer of filtering membrane such as

1. The endothelium of capillary membrane,
2. Basement membrane and
3. Endothelium of visceral layer of Bowmen capsule.

The glomerular filtration is called ultra filtration because minute particles are filtered, but the plasma proteins are not filtered due to larger molecular size than size of the slit pores. The composition glomerular filtrate is similar to that of plasma except in the absence of plasma proteins.

Glomerular filtrate rate GFR

The total quantity of filtrate formed in all the nephron of both the kidneys in the given unit of time is called glomerular filtrate rate. The normal value of glomerular filtrate rate is 125 ml / minute or about 180 litres / day.

Filtration Fraction

The fraction of the renal plasma which becomes the filtrate is called filtration fraction. It is the ratio between renal plasma flow and glomerular filtrate. It is expressed in percentage.

$$\begin{aligned}
 \text{They filtration fraction} &= \frac{\text{GFR}}{\text{Renal plasma flow}} \times 100 \\
 &= \frac{125 \text{ ml / minute} \times 100}{650 \text{ ml / minute}} \\
 &= 19.2\%
 \end{aligned}$$

The normal filtration fraction values from 15 to 20%

Pressure determining filtration:

Glomerular capillary pressure, colloidal osmotic pressure in the glomeruli and the hydrostatic pressure in the Bowman's capsule, which are determine the GFR. Among these pressures, the glomerular capillary pressure favours filtration the glomerular capillary pressure is about 60mm Hg and is highest capillary pressure in the body. The colloidal osmotic pressure and hydrostatic pressure oppose the filtration. The colloidal osmotic pressure exerted by plasma protein in the glomeruli. The plasma proteins are not filtered through the glomerular capillaries, so increased

concentration of proteins in glomerulus during filtration causes the development of colloidal osmotic pressure. It is about 25mmHg. Hydrostatic pressure in Bowman's capsule is exerted by the filtrate in Bowman's capsule during filtration. It is about 15mmHg.

Net filtration pressure:

The balance between pressure favoring filtration and pressures opposing filtration is called net filtration pressure. This is very essential for the maintenance of GFR so this is otherwise known as effective filtration pressure of essential filtration pressure.

The net filtration pressure = Glomerular capillary pressure – colloidal osmotic pressure + Hydrostatic pressure in Bowman's capsule

$$= 60 - (25+15) = 20\text{mmHg}$$

The normal net filtration pressure is about 20mmhg and it varies between 15 and 20mmHg

Factors regulating GFR:

Following are the various factors, which regulate or affect the GFR

1. Tubular glomerular feedback mechanism

This is the process in which the GFR is constantly regulated by means of feed back from renal tubule. The macula densa of juxtaglomerular apparatus is responsible for this. When the glomerular filtrate passes through the end portion of thick ascending segment of renal tubule, the macula densa detects the concentration of sodium chloride and accordingly alters the GFR. If the concentration of sodium chloride is more, macula densa causes constriction of afferent arteriole and filtration rate decreases, the constriction of afferent arteriole may be due to the secretion of thromboxane A₂ from macula densa.

2. Glomerular capillary pressure

The GFR is directly proportional to glomerular capillary pressure. When glomerular capillary pressure is increased the GFR is also increased, in turn depends upon the renal blood flow

3. Colloidal osmotic pressure

The GFR is inversely proportional to colloidal osmotic pressure exerted by protein. During dehydration or increased plasma protein level, colloidal osmotic pressure is more and GFR is reduced.

4. Hydrostatic pressure in Bowman's capsule

GFR is inversely proportional to this. The hydrostatic pressure in Bowman's capsule is increased in conditions like obstruction of urethra and oedema of kidney beneath renal capsule.

5. Renal blood flow

This is the most important factor necessary for glomerular filtration. And, the GFR is directly proportional to this.

6. Constriction of afferent arteriole

This reduces the blood flow to the glomerular capillaries and this in turn reduces GFR.

7. Constriction of efferent arteriole

If efferent arteriole is constricted; initially there is an increase in GFR.

8. Systemic arterial pressure

However, increase in mean arteriole pressure up to 180mmHg or reduction up to 60mmHg does not alter renal blood flow or GFR. This is due to auto regulatory mechanism. Variation in pressure above 180mmHg or below 60mmHg affects the renal blood flow and GFR because auto regulating mechanism fails beyond this range.

9. Sympathetic stimulation

The mild or moderate stimulation of sympathetic nerves does not cause any significant change either in renal blood flow or in GFR. This is due to auto regulation. Strong sympathetic stimulation causes severe constriction of the blood vessels; needs to increase filtration initially, but later decreases.

10. Surface area of capillary membrane

GFR is directly proportional to the surface area of the capillary membrane.

Tubular reabsorption

When the glomerular filtrate passes through the tubular portion of nephron, both quantitative and qualitative changes occur. Large quantity of water, electrolytes and other substances are reabsorbed by the tubular epithelial cells. The substances, which are reabsorbed, pass in to the interstitial fluid of renal medulla, and from here, the substances move in to the blood in peritubular capillaries. As the substances are taken back in to the blood, the entire process is called tubular reabsorption.

Selective reabsorption

The tubular cells of kidney selectively reabsorb the substances present in the glomerular filtrate, according to the needs of the body. So the tubular reabsorption is called the selective absorption. Depending upon the degree of reabsorption, the various substances are classified into three categories.

1) High threshold substances

The food substances like glucose, amino acid, acetoacetate ions and vitamins are completely reabsorbed do not appear in urine under normal condition. These substances appear in urine, only if their concentration in plasma is abnormally high or in renal diseases. So these substances are called high threshold substances.

2) Low threshold substances

The substances such of urea, uric acid and phosphate are reabsorbed to little extend. These substances appear in urine even under normal conditions. Such substances are known as low threshold substances.

3) Non threshold substances

The metabolic end products like creatinine are not at all reabsorbed and are excreted in urine irrespective of their plasma level. These substances are called non threshold substances.

Mechanism of reabsorption

The mechanisms involved in tubular reabsorption are of two types

1. Active reabsorption and
2. Passive reabsorption.

1. Active reabsorption

The movement of molecules is against the electrochemical gradient. This needs liberation of energy. And the energy is derived from ATP. The substances reabsorbed actively from the renal tubule are sodium, calcium, potassium, phosphates, sulphates, bicarbonates, glucose, amino acids, ascorbic acid, uric acid and ketone bodies.

2. Passive reabsorption

In this process, the movement of molecules is more along the electrochemical gradients. This process does not need energy, the substances reabsorbed by passive transport are chloride, urea, and water.

Site of reabsorption

Substances reabsorbed from proximal convoluted tubule. Glucose, amino acids, sodium, potassium, calcium, bicarbonates, chlorides, phosphates, uric acid and water are reabsorbed from proximal convoluted tubule. The substances reabsorbed from loop of Henle are sodium chloride. The substances reabsorbed from distal convoluted tubule are sodium, bicarbonate and water.

Tubular secretion

Some substances secrete in to the lumen from the peritubular capillaries through the tubular epithelial cells. These known as tubular secretion or tubular excretion.

1. Potassium is secreted actively by sodium – potassium pump in distal convoluted tubule and collecting duct.
2. Ammonia is secreted in the proximal convolute tubule.

3. Hydrogen ions are secreted in the proximal and distal convoluted tubules. Maximum hydrogen ion secreted in proximal tubule.

Thus by the process of glomerular filtration, selective reabsorption and tubular secretion urine is formed in the nephron. It is also concentrated by counter current mechanism and ADH. Finally it passes through ureter into the urinary bladder and is stored there until it is voided out.

URINARY CALCULUS

Urinary calculus is stone –like body composed of urinary salts bound together by a colloid matrix of organic material, it consist of nucleus around which concentric layers urinary salt are deposited . Urinary stones have afflicted humankind since antiquity, with earliest recorded example being bladder and kidney stones detected in Egyptian mummies dated to 4800 B C.

Until the 1980s, urinary stones were a major problem, with a significant proportion of patients requiring extensive surgical procedures and a sizable losing their kidney. The advent of extra corporeal techniques for stone destruction and refinements in endoscopic surgery, however have greatly decreased the morbidity associated with stone surgery.

EPIDEMOLOGY

INTRINSIC FACTORS

Hereditary

Several disorders that cause renal stones are hereditary .Familial renal tubular acidosis are associated with nephrolithiosis in almost 70% of patients. Cystinuria, Xanthinuria and Dehydroxyadenuria are disorders cause renal stone

Age and sex

The peak incidence of urinary calculus occurs in the twenties to forties. About three males are affected for every female because increased serum testosterone causes increased endogenous oxalate production in male, increased urinary citrate in women.

EXTRINSIC FACTORS

Geography

The prevalence of urinary calculi is higher in those who live in mountainous, desert, and tropical areas.

Climate and seasonal factors

Price and associates found that the incidence of urinary calculi was higher during the summer months. High temperatures increase perspiration, which may result in concentrated urine. This promotes increased urinary crystallization. Parry and Lister suggest that increased exposure of sun light cause increased production of 1, 25-dihydroxyvitamin D3 and increased urinary calcium excretion. This may cause higher incidence of urolithiasis in summer months.

Water Intake

Urine dilution by increased water intake may increase ion activity coefficients and hence urinary crystallization, water diuresis reduces the average time of residence of free crystal particles in urine dilutes components of urine that may form crystals. Minerals contents of water may contribute to cause stone disease. (E.g. sodium chlorides) Zinc is an inhibitor of calcium crystallization.

Diet

Ingestion of excessive amounts of purines, oxalates, calcium, phosphates, and other elements often results in excessive excretion of these components in urine causes increased incidence of calculi.

Occupation

Lonsdale (1968b,c) indicated that urinary calculi are much more likely to be found in individuals who have sedentary occupations. The highest were found in cooks and engineering room personnel. The risk of calcium oxalate and uric acid stones formation in Astronauts because of hypercalciuria, hypocitraturia, decreased pH, and lower urinary volumes.

PHYSICAL CHEMISTRY

Urinary stones do not occur unless crystals of the offending substance form in urine. For crystals to occur, the urine should be supersaturated with the salt in consideration. Increases in urinary excretion of chemicals that constitute the crystals result in an increase in potential for crystallization. Urine does not need to be continuously supersaturated

for crystals to form or grow: Intermittent super saturations, as is seen during of dehydration or after meals, is sufficient. Crystallizing potential for calcium oxalate is related not so much to the concentration of calcium or oxalate in urine but to chemical activity of ions in solution. The compounds such as citrate and phosphate form complexes with calcium, and elements such as magnesium and sodium form complexes with oxalate, effectively reducing the free ionic concentration of each.

Urinary crystals can be seen in most urine specimens, particularly after storage, yet most individuals do not form stones. Stone formers as a group excrete larger crystals and crystals aggregates than healthy individuals. Normal subjects have inhibitors of crystal formation, growth, and aggregation in urine such as low-molecular-weight compounds such as citrate and pyrophosphate and larger molecules, such as glycosaminoglycans, nephrocalcin, and Tamm – Horsfall protein. Urine from patients with recurrent calcium and oxalate stones tends to have higher calcium and oxalate saturation and lower inhibitors than urine from patients without stones and a mathematically derived saturation - inhibition index has been reported to discriminate between the groups with better than 90% accuracy.

Free crystals formed within the kidney do not have the ability to grow to a size large enough to occlude a collecting duct and, form a stone in a free – flowing urinary system. Crystal aggregation and retention within the kidney are prerequisites for urinary crystals to be converted to urinary calculi. Crystal aggregation is enhanced in individual who lack inhibitors of aggregation. The urinary glycoproteins nephrocalcin and Tamm-Horsfall protein are potent inhibitors of crystal aggregation in simple solutions, whereas citrate and magnesium are inhibitors of crystal growth.

Anatomical abnormalities, such as medullary sponge kidney or ureteropelvic junction obstruction, or increased “stickiness” of the tubular epithelium can predispose to increased crystal retention. Urate and calcium oxalate crystals anchor to surfaces of cultured renal epithelial cells and may adhere in vivo to tubular cells or urothelium. Although not

proven, bacterial infection may promote calcium oxalate stone formation by increasing urinary matrix, which, in turn, promotes crystal adherence. Finally, altered transport of calcium and oxalate by renal epithelial cells may result in intracellular or interstitial crystallization. These are retained in the kidney and can become the nidus for stone formation.

TYPES OF CALCULUS

CALCIUM OXALATE STONES

This is most common type of stones. 39% of patients are calcium oxalate type stone. 14% of patients are combined with calcium phosphate. They are irregular in shape and covered with sharp projection, which tend to cause bleeding. The surface of the stone is discoloured by pigments of altered blood. It is very hard and absorbs X-rays well.

PATHOPHYSIOLOGY OF STONE FORMATION

I. HYPERCALCIURIA

Between 30% and 60% of all patients with calcium oxalate kidney stones have increased urinary calcium excretion in the absence of raised serum calcium levels. Hypercalciuria has been defined as the excretion of greater than 4mg calcium per kg body weight per day or greater than 7 mmol in men and 6 mmol in women (parks and Coe, 1986). Final definition is the excretion of urinary calcium of greater than 0.11 mg/100 ml of glomerular filtrate. Hypercalciuric nephrolithiasis suffer from multiple disturbances in renal tubular function a disturbance in phosphate transport, and accelerated 1, 25 – dihydroxyvitamin D3 synthesis, resulting in increased intestinal calcium absorption.

Hypercalciuria has three types, such as

1. Absorptive hypercalciuria,
2. Renal hypercalciuria
3. Resorptive hypercalciuria,

1. Absorptive hypercalciuria

In absorptive hypercalciuria, the primary abnormality is increased calcium absorption. In absorptive hypercalciuria type 1, intestinal hyperabsorption of calcium exists, whether or not the patient is on a calcium-restricted diet. Intestinal magnesium absorption is normal in patients with absorptive hypercalciuria, but oxalate absorption is increased. Vitamin D increases calcium and magnesium, but not oxalate, absorption in both the jejunum and ileum. Absorptive hypercalciuria type 2 is a variant of this disorder wherein patients exhibit increased urinary calcium excretion while on their normal diet but normal calcium excretion on a low-calcium, low-sodium diet. In the final subcategory, absorptive hypercalciuria type 3, the serum phosphate is low, suggesting that the increased intestinal calcium absorption is the result of stimulation in vitamin D production as the result of the lowered serum phosphate.

2. Renal hypercalciuria

In this condition, the underlying abnormality is a primary renal wasting of calcium. The consequent reduction in circulating serum calcium stimulates PTH production. Two facts must be stressed; first, intestinal absorption of calcium is increased in both absorptive and renal hypercalciuria; second, parathyroid function is suppressed in absorptive hypercalciuria but stimulated in renal hypercalciuria. These two criteria elevated fasting urinary calcium levels and stimulated parathyroid function serve to distinguish renal from absorptive hypercalciuria.

3. Resorptive hypercalciuria

This syndrome is synonymous with subtle hyperparathyroidism. Hypercalciuria results from excessive PTH dependent bone resorption as well as enhanced intestinal absorption of calcium.

4. Idiopathic hypercalciuria

The syndrome of renal phosphate leak, elevated 1, 25 dihydroxyvitamin D₃, and absorptive hypercalciuria has been demonstrated in members of a Bedouin tribe in which intermarriage is common. Idiopathic

hyper calciuria may be inherited as an autosomal trait, although the pattern can reflect polygenic control of calcium excretion as well.

II. HYPERCALCAEMIA NEPHROLITHIOSIS

CAUSES

1. Primary hyperparathyroidism

These patients are between 39% and 78% with early presented of renal calculi. These patients with higher level of 1, 25-dihydroxyvitamin D3 and greater calcium absorption tend to have renal stones.

2. Malignancy associated hypercalcemia

Malignancy –associated hyper calciuria is an exceedingly rare cause of renal stones. The most common cause of malignancy- associated hypercalcaemia, even in patients with skeletal metastasis, is production by the tumour of a bone resorpting substance called PTH- related polypeptide.

3. Sarcoidosis and other granulomatous diseases

The sarcoid granuloma produces 1, 25-dihydroxyvitamin D3, it causing increased calcium absorption, hypercalcaemia, and hypercalciuria.

4. Hyperthyroidism

About 5% to 10% of patients with hyperthyroidism develop hypercalcimia. Hypercalcaemia and hypercalciuria result from a stimulation of bone resorption mediated by thyroxine and tri-iodothyronine.

5. Glucocorticoid – induced hypercalcemia

Glucocorticoid excess leads to increased bone resorption, decreased bone formation, and osteopenia. Glucocorticoid also has direct stimulatory effect on parathyroid gland.

6. Pheochromocytoma

Hypercalcaemia when seen in patients with pheochromocytoma, occur most often in patient with multiple endocrine neoplasma type 2, in which primary hyperparathyroidism, medulary carcinoma of thyroid, adrenal gland tumour coexit.

7. Immobilization

Prolonged bed rest can lead to hypercalcaemia, as the result of increased bone turnover. Hypercalcaemia is seen most often when another condition, such as Paget's disease- with accelerated bone turnover-primary hyperthyroidism, or malignancy, coexists in an immobilized patient.

III. HYPEROXALURIA

CAUSES:

Primary hyperoxaluria is a rare genetic disorder resulting from increased hepatic production of oxalate. Enteric hyperoxaluria occurs in patients with short bowel syndrome or malabsorption. Finally, a group of patients with recurrent idiopathic calcium oxalate lithiasis exhibits mild hyperoxaluria or increased transport of oxalate by red blood cells.

1. Primary hyperoxaluria

Two types of primary hyperoxaluria exist. Primary hyperoxaluria type 1 is an autosomal recessive inborn error of metabolism characterized by nephrocalcinosis and oxalosis. The diseases are characterized by increased urinary excretion of oxalic, glycolic, and glyoxylic acids. Primary hyperoxaluria type I is due to a defect of the enzyme alanine-glyoxylate aminotransferase (AGT) in the liver.

In normal human liver, AGT catalyzes the transamination or detoxification of glyoxylate to glycine, a function that it can perform only if it is located in the peroxisome. Its deficiency in primary hyperoxaluria results in glyoxylate's being oxidized to oxalate.

Primary hyperoxaluria type II or L-glyceric aciduria is a much rarer variant of the disease. Deficiencies of the hepatic enzymes D-glycerate dehydrogenase and glyoxylate reductase lead to increases in urinary oxalate and glycerate excretion.

2. Enteric hyperoxaluria

Ingested oxalate is absorbed through the stomach and the colon. Malabsorption from any cause, including small bowel resection, intrinsic

disease, or jejunioileal bypass, increases the colonic permeability of oxalate as the result of exposure of the colonic epithelium to bile salts. Furthermore, loss of calcium in the feces results in the presence of less calcium in the intestinal lumen, allowing oxalate to exist in a soluble form. The hyperoxaluria from small bowel malabsorption often exceeds 1 mmol/day and causes recurrent nephrolithiasis, nephrocalcinosis, and renal oxalate deposition.

3. Mild metabolic hyperoxaluria

Mild hyperoxaluria is at least as important a factor in the pathogenesis of idiopathic calcium oxalate stones as hypercalciuria. Baggio and colleagues are found an increase in oxalate self exchange across red blood cell membrane in 79% of patients with idiopathic calcium oxalate stones. The oxalate absorption was increased with increased calcium absorption. Dietary restriction of oxalate results in decreased oxalate excretion.

IV. HYPERURICOSURIA

Uric acid promotes calcium oxalate crystallization by facilitating the formation nuclei. Sodium acid urate may produce calcium oxalate stone disease by nullifying the effectiveness of naturally occurring inhibitors of calcium oxalate crystal growth. Excessive dietary intake of purine is main cause of hyperuricosuria. Between 80% and 90% of patients with hyperuricosuria nephrolithiasis are men. Patients with mixed with uric acid and oxalate stones have lower urinary pH than patients with pure calcium oxalate stones.

V. HYPOCITRATURIA

Hypocitraturia has been reported in 15% to 63% of patients with stones. Urinary citrate is normally greater in women than in men. Hypocitraturia define as citrate excretion of less than 0.60 mmol (115mg) in men and 1.03 mmol (200mg) in women. Acidosis is probably the most important etiologic factors in hypocitraturia. In patients with inflammatory bowel disease and chronic diarrhea, intestinal alkali loss results in metabolic acidosis. Thiazide- induced hypokalaemia and intracellular

acidosis are other cause of decreased urinary citrate excretion. A diet rich in animal protein may produce an acid load. Strenuous physical exercise and sodium intake can likewise produce hypocitraturia. Urinary tract infection with bacteria degrading citrate lowers urinary citrate excretion.

The primary mechanism of action of citrate is as a complexing agent for calcium. Calcium citrate complexes are considerably more soluble than calcium oxalate. Citrate inhibits the spontaneous nucleation of calcium oxalate, crystal growth and aggregation of calcium oxalate and phosphate.

VI. HYPOMAGNESURIA

Many experimental studies have suggested that administration of magnesium salts prevents stones disease. The most common cause of hypomagnesuria is inflammatory bowel disease associated with malabsorption. Most patients with hypomagnesuria also have hypocitraturia.

VII. SEX HORMONES AND RENAL STONES

Calcium oxalate renal stones occur much more frequently in men than in women because increased serum testosterone levels resulted in increased endogenous oxalates production by liver and increased intestinal absorption of calcium in men, increased urinary citrate concentration in urine in women.

CALCIUM PHOSPHATE STONES

Calcium phosphate stones composed predominantly around 10% of stones of renal origin. Although some amount of calcium phosphate is often found in calcium oxalate calculi, pure calcium phosphate stone are rare. It is usually smooth and dirty white, and easy to see on radiopaqhic film.

Causes:

Renal tubular acidosis is common cause for this type of stone. Up to 70% of adults with renal tubular acidosis have kidney stones. This stone

may be seen with oxalate and struvite stones. Stone formation typically occurs in papillary tips and in medulla. Stone formation is result of hypercalciuria, hypocitraturia, and increased urinary pH. Hypercalciuria is result of systemic acidosis on bone demineralisation and secondary hyperthyroidism. Hypocitraturia result from a primary defect in renal tubular citrate transport, again, the result of metabolic acidosis. Hypocitraturia is probably the most important metabolic factor for stone formation in patient with renal tubular acidosis. The defect in proximal tubular bicarbonate resorption is associated with this type of stone.

Diagnosis: the patient has hypokalaemia, hyper chloremia, metabolic acidosis and urinary pH of 5.5.

URIC ACID STONE

These are hard, smooth and often multiple. Their colour varies from yellow to reddish brown. The pure uric acid stone are radiolucent most of stone with calcium so they radiolusant shadow.

Causes:

The principal cause of uric acid crystallization is supersaturation of urine with respect to undissociated uric acid. The normal 24-hour urinary uric acid excretion is between 500 and 600mg per litre of urine. Patient with uric acid stone often have prolonged periods of acidity in urine. The pH of urine in patient with uric acid stones was 5.5 ± 0.4 as compared with 6.0 ± 0.4 in patients who form calcium oxalate stones. Three factors are involved in uric acid urolithiosis. First, patients tend to excrete excessively acid urine at relatively fixed, low urinary pH. Second, they may absorb, produce, or excrete more uric acid than patients without gout or uric acid stones. Third, urinary volume is diminished in these patients. The combination of these factors is ideal for the crystallization of uric acid in the urine. The frequency of uric acid stones in gout is about 20%. Myeloproliferative disorders such as acute leukemia are an important cause of severe hyperuricosuria, particularly in childhood.

STRUVITE STONES (INFECTION STONES)

The stone is composed of magnesium, ammonium, and phosphate, mixed with carbonate.

Pathogenesis

Two conditions must coexist for the crystallization of struvite- a urine pH of 7.2 or above and the presence of ammonia in the urine. A second mechanism by which bacterial infection may induce stone formation is by increasing crystal adherence. Urease producing bacteria hydrolyze urea to carbondioxide and ammonium molecules. Two molecules of ammonia are produced from one molecule of urea; neutralization of the base is incomplete. As a result of this, the urinary pH rises.

Clinical presentation

Struvite calculus accounts are the majority of staghorn stones observed in most countries. They can grow quite large and may fill the collecting system. Struvite stones can form on a nidus of calcium oxalate stones and can grow quite rapidly. Most infection stones are radiopaque, but poorly mineralized matrix stones are faintly radiopaque or radiolucent. Women, perhaps because of their increased susceptibility to urinary tract infections, are more commonly affected than men. The presence of a foreign body in the urinary tract and neurogenic bladder are main causes for struvite calculi.

Patients with struvite stones may present acutely with fever, loin pain, dysuria, frequency, and hematuria. Metabolic abnormalities are present in patients with mixed calciumoxalate and struvite stones but not in patients with pure struvite stones.

CYSTINE STONES

They are uncommon Cystine stones account for about 1% of all urinary calculi in the United States and occur only in patients who have cystinuria. Cystinuria is an autosomal recessive disorder of transmembrane cystine transport manifested in the intestine and in the kidney. Cystine stones are radiopaque, although less so than calcium

oxalated stones. The radiopacity is the result of the disulfide bond in cystine. The stones are hexagonal, translucent white colour appear in acid urine and it is yellowish or pinkish color when first removed but greenish hue color when exposed to air and have a waxy appearance and very hard. They are often multiple, are large, and may form staghorns. Cystinuria can cause renal stones in childhood, but the peak of clinical expression is in the second and third decades. The cystine stones form because cystine is poorly soluble within the range of normal urinary pH.

XANTHINE STONES

These are extremely rare. They are smooth and round, brick –red in color and a radiolucent. Xanthinuria is an inborn error of metabolism, inherited as an autosomal recessive trait and characterized by a deficiency of xanthine oxidase. Serum uric acid levels are low, averaging less than 1.5 mg/dl. Serum and urine levels of xanthine and hypoxanthine are significantly increased. Xanthine stones develop because xanthine is less soluble than hypoxanthine.

MATRIX CALCULI

Matrix calculi are found predominantly in individuals with infections caused by urease-producing organisms. Proteus species are especially likely to be associated with matrix calculi. These stone composed of coagulated mucoids with little crystalline component. They are radiolucent and may be confused with uric acid calculi. Their association with alkaline urinary tract infection, however, usually assists in making a presumptive diagnosis because uric acid calculi are usually formed in acidic, sterile urine. Stones composed of β 2-microglobulin, a protein that is filtered and appears in the urine, may form in the kidneys of uremic patients.

CLINICAL PRESENTATION

Acute stone episode

A urinary calculus usually presents with an acute episode of renal or ureteral colic as the result of a stone obstructing the urinary tract. There

are five locations where stones can be impacted in the urinary tract. First, stones may become impacted in calyx of the upper urinary tract. Symptoms are abdominal distended and painful and create hematuria. The second area in which a calculus may become impacted is the ureteropelvic junction. A third area of impaction is at or near the pelvic brim, where the ureter begins to arch over the iliac vessels posteriorly into the true pelvis. The fourth area, especially in females, is the posterior pelvis, where the ureter is crossed anteriorly by the pelvic blood vessels and by the broad ligament; finally, the most constricted area through which the urinary calculus must pass is the ureterovesical junction, which is the most common site of impaction. Other impaction is urinary bladder and urethra.

Renal colic

The partially obstructing, continuously moving calculus appears to create the greatest amount of colic. As the stone moves to the midureter, pain generally tends to radiate to the lateral flank and abdominal area. When ureteral stones are near the bladder, patients often develop the symptoms of urinary frequency and urgency. Because of the autonomic nervous system transmits visceral pain. Nausea and vomiting are commonly associated with renal colic.

Physical signs

Pulse rate and blood pressure may be elevated because of pain and agitation. Examination of the abdominal reveals moderate deep tenderness on palpation over the location of the calculus and the area of the loin.

INVESTIGATION

Blood

Calcium, Phosphate, Uric acid, Urea and Electrolytes
Parathyroid hormone.

Urine

Protein, Glucose, Blood, and Amino acid.

24-hr Urine examination

Urea, Creatinine clearance, Sodium, Calcium, Oxalate, and Uric acid.

Urine culture**Stone analysis: Chemical composition****Plain abdominal radiograph****Intravenous Urogram****Computed Tomography****Ultrasonography****CTscan****Modern methods of stone removal for Kidney stones:**

Percutaneous nephrolithotomy, spiral CT, extracorporeal shock-wave lithotripsy. Open surgery for renal calculi are Pyelolithotomy, extended pyelolithotomy, nephrolithotomy.

Treatment for Ureter calculi:

Endoscopic stone removal, ureteroscopic stone removal and lithotripsy in situ. Open surgery is ureterolithotomy.

MATERIALS AND METHODS

1. AIMS

a) Primary Aim

To estimate the efficacy of *Kalladaippu thool* in the treatment of *Kalladaippu*.

b) Secondary Aim

To find out the side-effects of the trial drug, if any.

2. POPULATION AND SAMPLE

The population consists of all patients with *Kalladaippu* diagnosed by clinical symptoms and Ultrasonography satisfying the inclusion and exclusion criteria mentioned below. The sample consists of *Kalladaippu* patients attending the IPD/OPD of Ayothidoss Pandithar Hospital of the National Institute of Siddha, Chennai – 47.

3. SAMPLE SIZE

The trial size will be 30 patients.

4. INCLUSION CRITERIA

1. *Vatha, Pittha* and *Khaba kalladaippu*.
2. Aged between 20 and 60 years.
3. Willing to be admitted in the hospital for 8 weeks or willing to attend the O.P.D once a week for 8 weeks.

5. EXCLUSION CRITERIA

1. Severe infection
2. Kidney diseases
3. Diabetes insipitus
4. Pregnancy

6. WITHDRAWAL CRITERIA

1. Obstructive anuria
2. Severe haematuria
3. Severe pain

7. TRIAL DRUG AND DURATION

Kalladaippu thool (powder) 5 g with 50 ml of raddish juice, twice a day, for 8 weeks.

8. CLINICAL ASSESSMENT AND INVESTIGATION

a) CLINICAL ASSESSMENT

IN SIDDHA ASPECT

1. ***Vatha Kalladaippu***: Retention of urine, lower abdominal pain with distention, pain with swelling of penis, agonizing pain, breathlessness and excretion of urine with mucus.

2. ***Pittha Kalladaippu***: Stone blocked in urinary tract followed by oliguria, pain in urethra like an insertion of burning iron bar, sweating all over the body, anuria, agonizing pain, blood stained calculus excreted in urine with piercing and boring pain and hyperalgia.

3. ***Khaba Kalladaippu***: Abdominal distention with pain like a bow, pain in all joints of upper and lower limbs, lumbar region, hip region and penis, sweating all over body and white colored stone excreted in urine.

b) INVESTIGATION

1. BLOOD

TC (cells/cumm), DC (%), Hb (g %), ESR (mm/hr), Blood Sugar (R) (mg %), Blood Urea (mg %), Serum Creatinine (mg %) and Serum Cholesterol (mg%).

2. URINE

Reaction (pH), Albumin, Sugar and Deposits.

3. MOTION

Ova, Cyst, and Occult blood.

4. ULTRA SONOGRAPHY

5. IN SIDDHA SYSTEM

Envagai thervukal: Naa, Niram, Mozhi, Vizhi, Malam, Moothiram (neerkuri and neikuri), Sparism and Naadi.

9. CONDUCT

Kalladaippu patients satisfying the inclusion and exclusion criteria will be admitted to the trial. Informed consent will be obtained from the patients. Lab investigation will be carried out before treatment and at the end of the treatment.

For the I.P. patients trial drug will be administered by the doctor and clinical assessment will be done weekly. The trial drug will be issued to the O.P. patients for 7 days at a time. Patients will be asked to come for clinical assessment once in 7 days. Also, they will be asked to bring back unconsumed drug at each visit and return them.

11. FORMS

Form I: Selection proforma - Used before admission.

Form II: Assessment proforma – Used once in a week during treatment.

12. ANALYSIS

Paired X^2 – test for changes in signs and symptoms.

Paired t-test for changes in objectives parameters.

OBSERVATION AND RESULTS

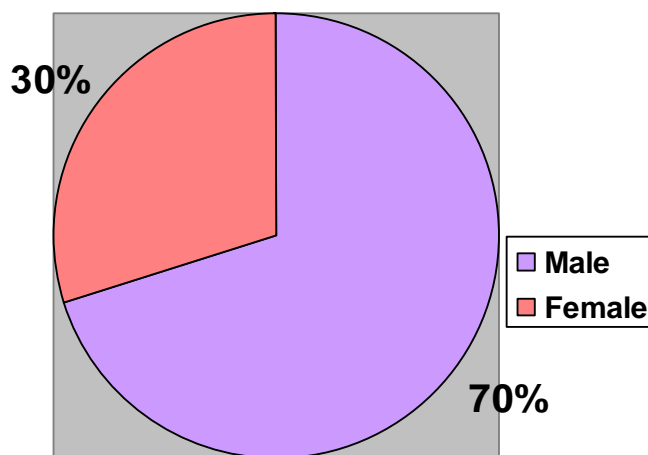
30 cases of kalladaippu were treated with kalladaippu thool 5gms with 50ml raddish juice, two times for 8 weeks in NIS. The observation are tabulated, the patient who had positive ultrasonography, positive urine examination finding of crystals, R B C, albumin and puscells were included in this group. Tables were formed with reference of age, sex, diet history and personal habits seasonal incidence, distribution of land, clinical laboratory investigation and diagnosis.

DISTRIBUTION OF CASES BY GENDER

TABLE: 1

Gender	Cases	
	No	%
Male	21	70.0
Female	9	30.0
Total	30	100.0

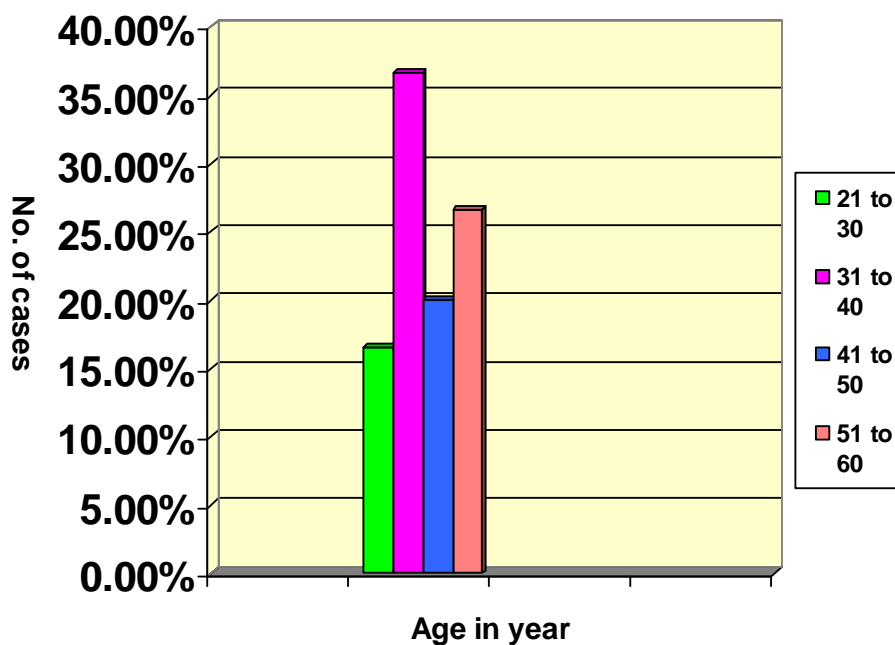
Distribution of cases by gender



DISTRIBUTION OF CASES BY AGE
TABLE: 2

Age(yr)	Cases	
	No	%
21 to 30	5	16.6
31 to 40	11	36.6
41 to 50	6	20.0
51 to 60	8	26.6
Total	30	100.0

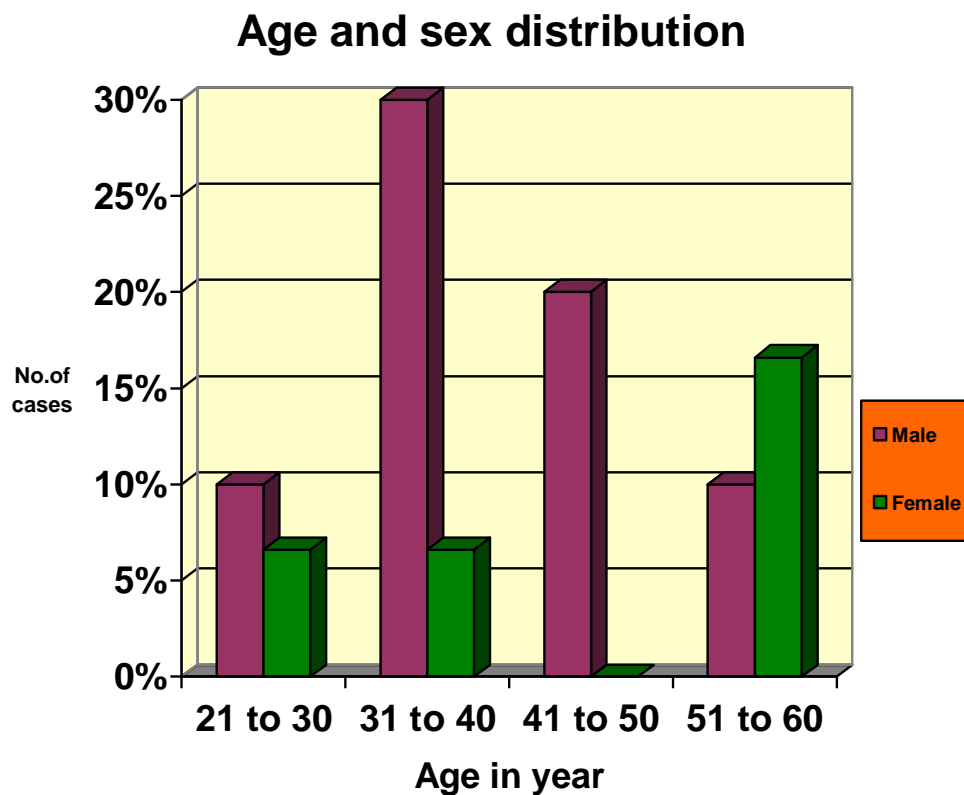
Distribution of cases by age



DISTRIBUTION OF CASES BY AGE AND SEX

TABLE: 3

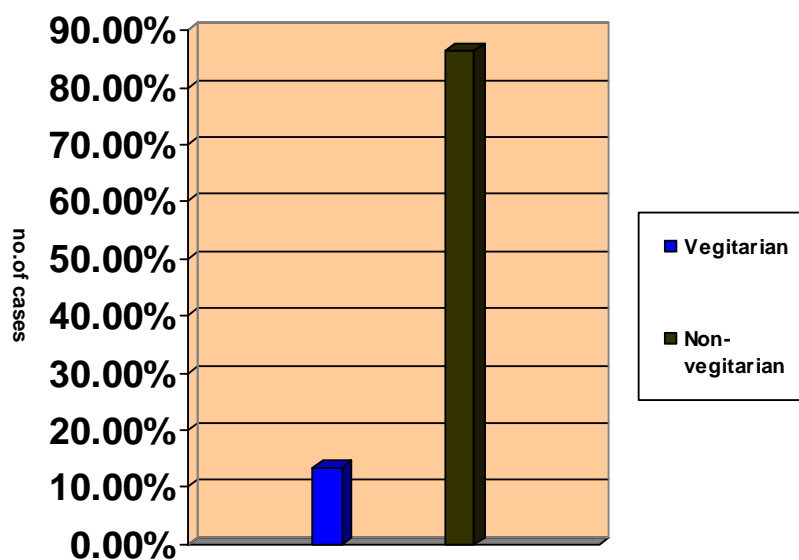
Age(yr)	Cases					
	Male No	%	Female No	%	Total	%
20 to 30	3	10.0	2	6.6	5	16.6
31 to 40	9	30.0	2	6.6	11	36.6
41 to 50	6	20.0	0	0	6	20.0
51 to 60	3	10.0	5	16.6	8	26.6
Total	21	70.0	9	30.0	30	100.0



DISTRIBUTION OF CASES BY FOOD HABITS
TABLE: 4

Food habits	Cases	
	No	%
Vegetarian	4	13.3
Non vegetarian	26	86.6
Total	30	100.0

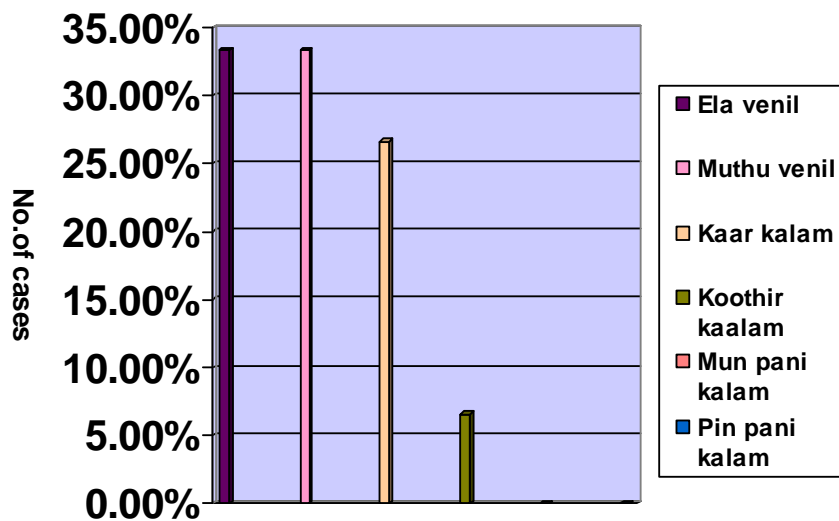
Distribution among Food habit



DISTRIBUTION OF CASES BY SEASONAL
TABLE: 5

Paruvakalam	Cases	
	No	%
Elavenil kalam	10	33.3
Muthuvenil kalam	10	33.3
Kaar kalam	8	26.6
Koothir kalam	2	6.6
Munpani kalam	0	0
Pinpani kalam	0	0
Total	30	100.0

Distrbution among paruva kalam

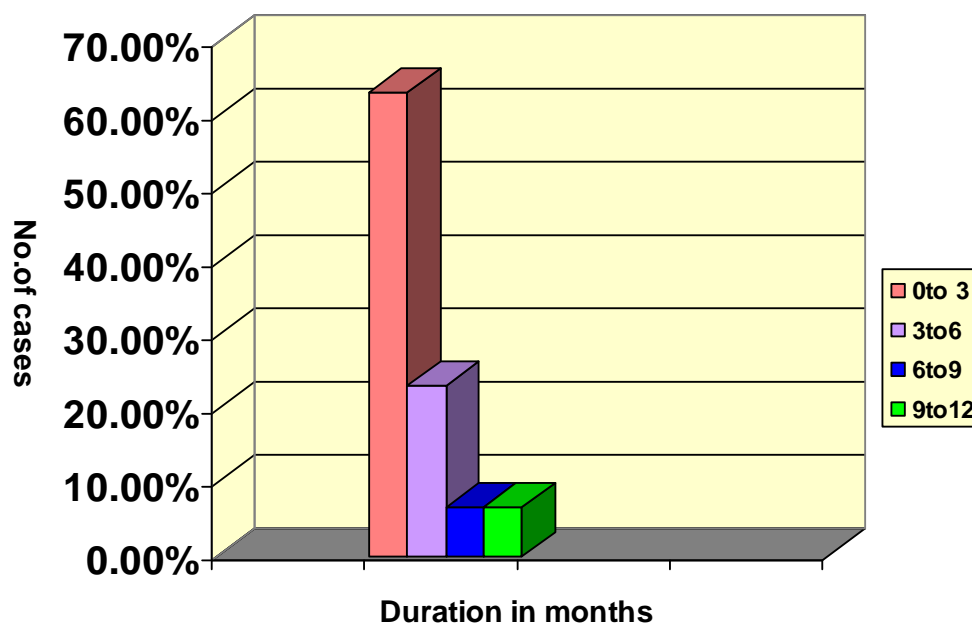


DISTRIBUTION OF CASES BY DURATION OF DISEASE

TABLE: 6

Duration in months	Cases No	%
0 to 3	19	63.3
3 to 6	7	23.3
6 to 9	2	6.6
Total	30	100.0

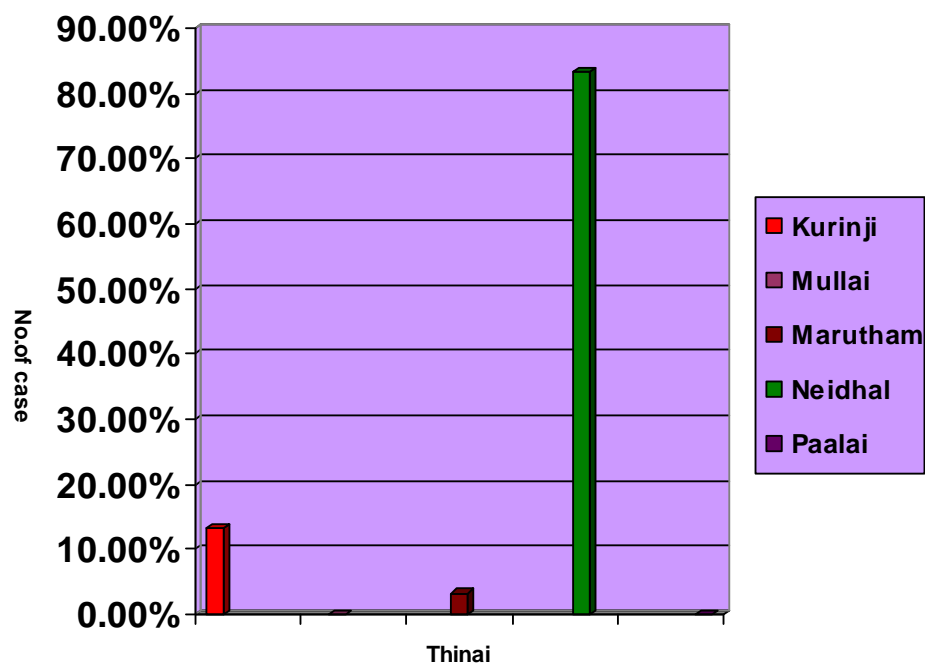
Distribution among duration of illness



DISTRIBUTION OF CASES BY THINAI
TABLE: 7

Thinai (Land)	Cases	
	No	%
Kurinchi (Hill)	4	13.3
Mullai (Forest)	0	0
Marutham (Fertile)	1	3.3
Neithal (Costal)	25	83.3
Paalai (Desert)	0	0
Total	30	100.0

Distribution among Thinai

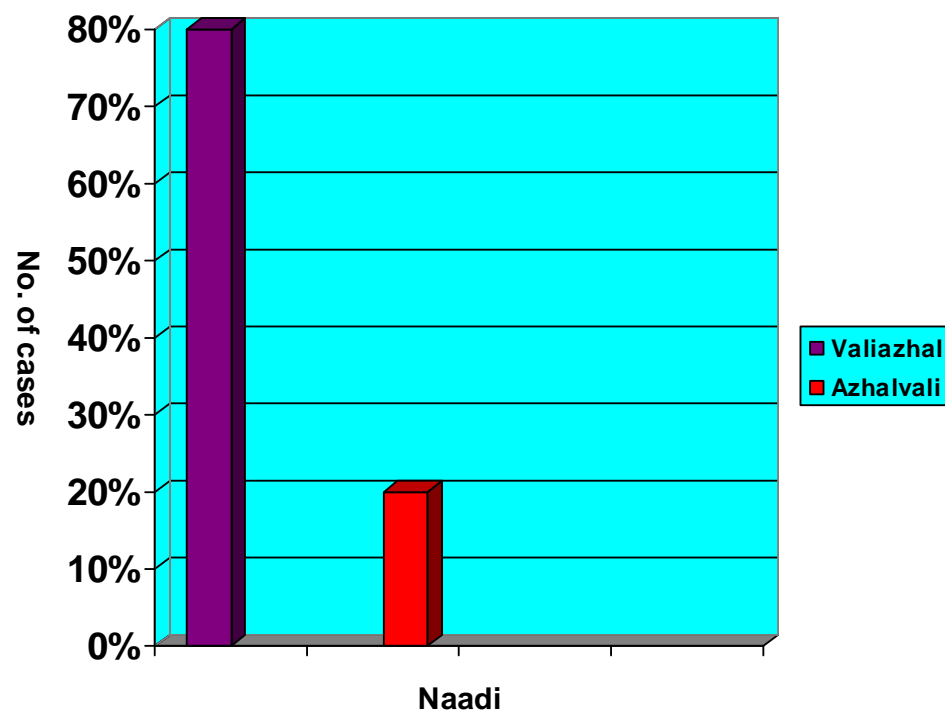


DISTRIBUTION OF CASES BY YAKKAI

TABLE: 8

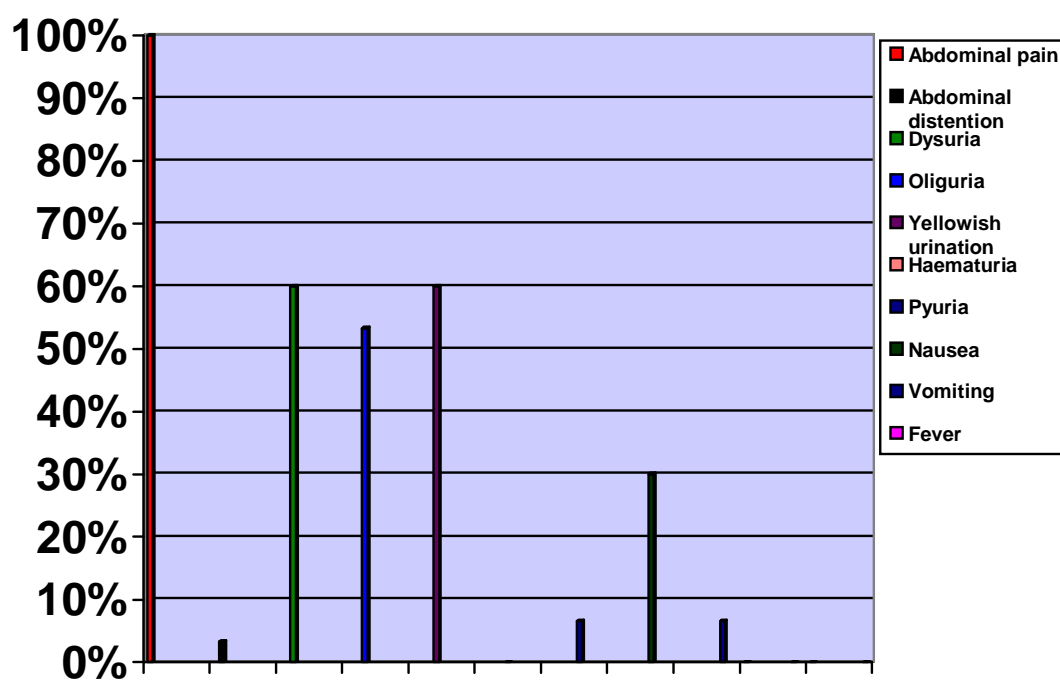
Yakkai	Cases	
	No	%
Valiazhal	24	80.0
Azhalvali	6	20.0
Total	30	100.0

Distribution of cases by naadi



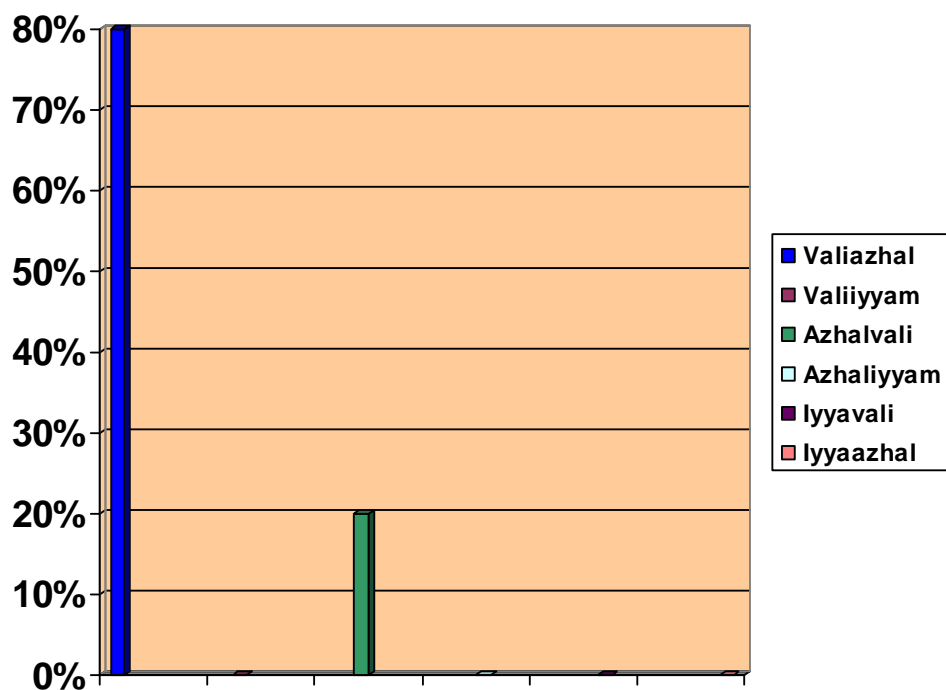
DISTRIBUTION OF CASES BY CLINICAL FEATURES
TABLE: 9

S.NO	Clinical features	Cases	
		No	%
1	Abdominal pain	30	100.0
2	Abdominal distention	1	03.3
3	Dysuria	18	60.0
4	Oliguria	16	53.3
5	Yellowish urination	18	60.0
6	Haematuria	0	0
7	Pyuria	2	06.6
8	Nausea	9	30.0
9	Vomiting	2	06.6



DISTRIBUTION OF CASES BY NAADI
TABLE: 10

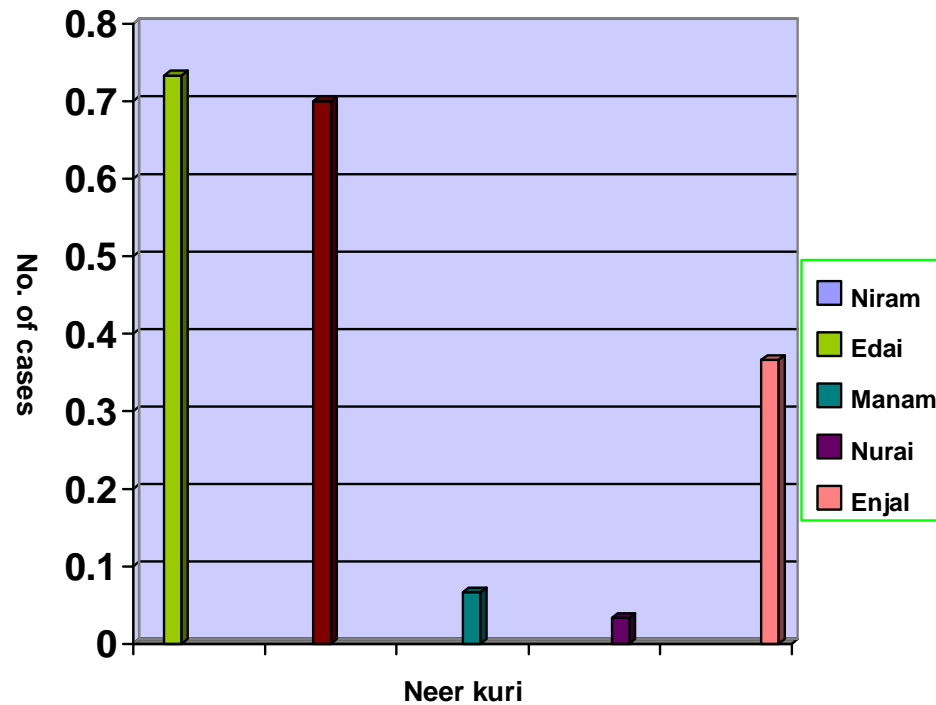
Naadi	Cases	
	No	%
Vali azhal	24	80.0
Vali iyyam	0	0
Azhal vali	6	20.0
Azhal iyyam	0	0
Iyya vali	0	0
Iyua Azhal	0	0
Total	30	100.0



DISTRIBUTION OF CASES BY NEERKURI
TABLE: 11

S.NO	Neerkuri	Cases	
		No	%
1	Niram Yellowish	22	73.33
2	Edai Oliguria	21	70.00
3	Manam Tamarind	2	06.66
4	Nurai	1	03.33
5	Enjal	11	36.66

Distribution of cases among neerkuri

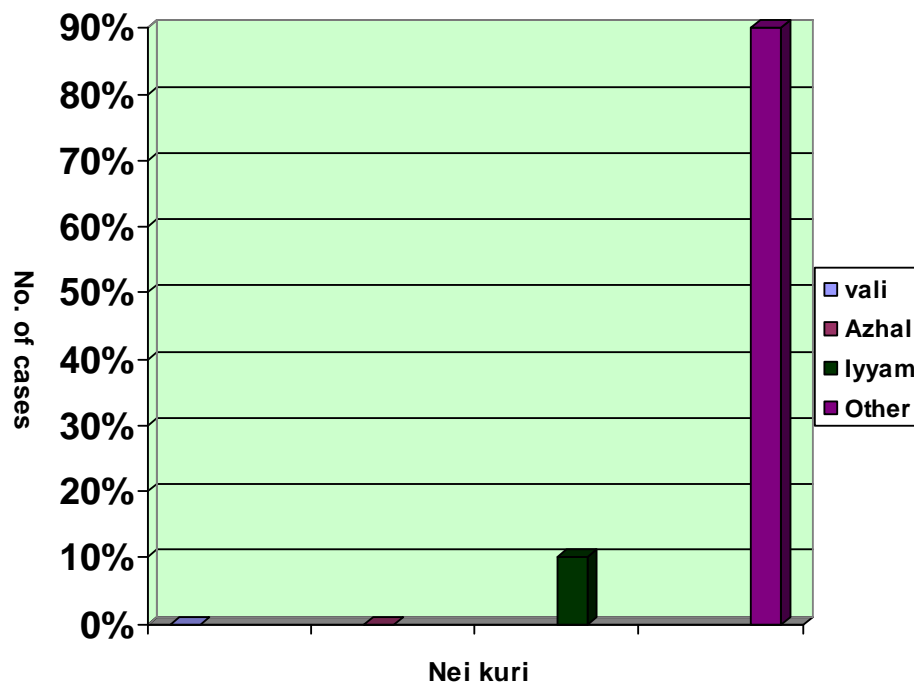


DISTRIBUTION OF CASES BY NEERKURI

TABLE: 12

Neikuri	Cases	
	No	%
Vali	0	0
Azhal	0	0
Iyyam	3	10.0
Thontham	0	0
Other Slowly spread	27	90.0
Total	30	100.0

Distribution of cases among neikuri

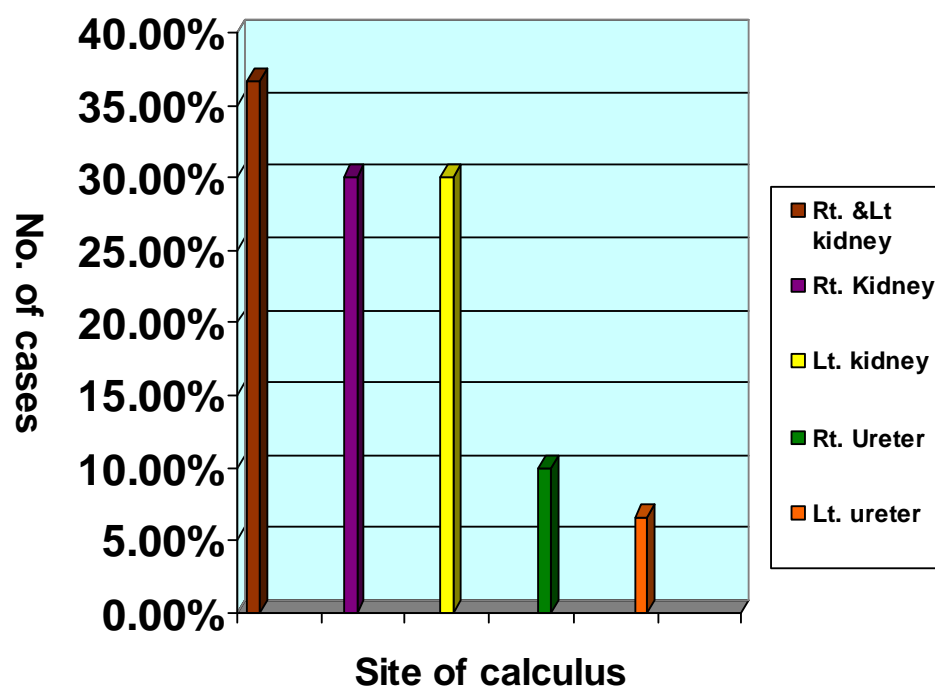


DISTRIBUTION OF CALCULI IN URINARY SYSTEM

TABLE: 13

S .NO	Site	No	%
1	Both kidneys	11	36.6
2	Right kidney	9	30.0
3	Left kidney	9	30.0
4	Right ureter	3	10.0
5	Left ureter	2	6.6

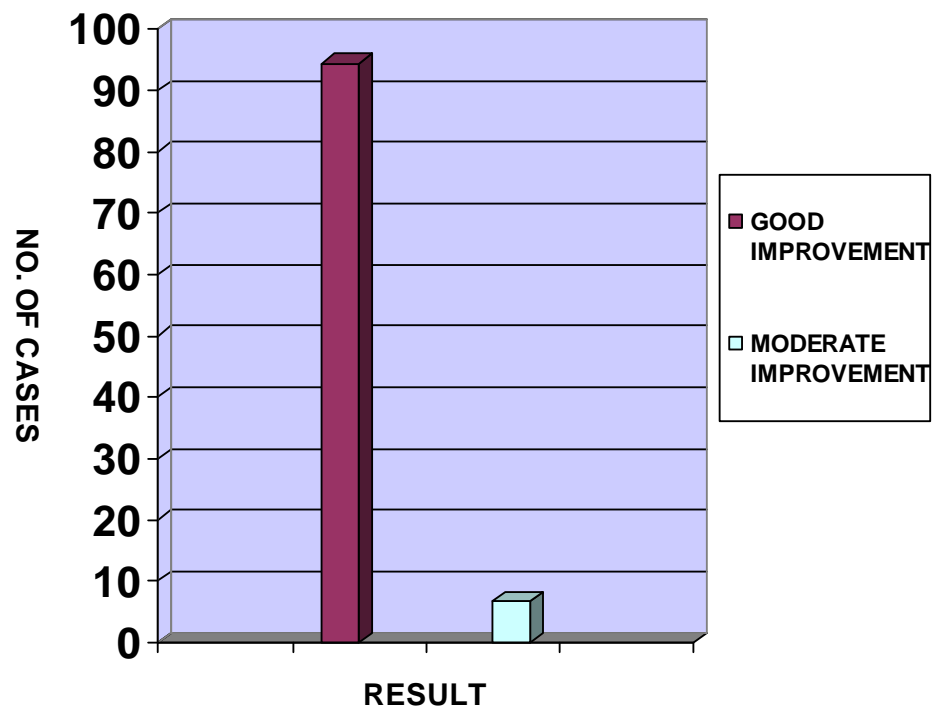
Distribution of cases among site of calculus



RESULT OF CLINICAL FEATURES AFTER TREATMENT
TABLE: 14

RESULT	NO. CASES	%
GOOD IMPROVEMENT	28	93.3
MODERATE IMPROVEMENT	2	6.6

RESULT AMONG CASES



REPORTS OF URINARY INVESTIGATION

Table: 15

S. NO	OP.NO	URINE INVESTIGATION							
		BEFORE TREATMENT				AFTER TREATMENT			
		ALBU MIN	DEPOSIT			ALBU MIN	DEPOSIT		
			PUS CELLS	EPI CELLS	CRYS TALLS		PUS CELLS	EPI CELLS	CRYS TALLS
1	R. 8855	Nil	2 to 4	2 to 4	Nil	Nil	1 to 2	1 to 2	Nil
2	S. 359	Nil	1 to 2	1 to 2	Oxalate	Nil	2 to 3	1 to 2	Nil
3	S. 511	Nil	4 to 5	2 to 4	Nil	Nil	3 to 4	1 to 2	Nil
4	S. 2319	Nil	2 to 4	1 to 2	Oxalate	Nil	1 to 2	1 to 2	Nil
5	S. 3568	Nil	2 to 4	2 to 4	Nil	Nil	1 to 2	1 to 2	Nil
6	S. 4030	Trace	7 to 9	1 to 3	Nil	Nil	3 to 5	1 to 2	Nil
7	S. 4356	Nil	4 to 6	2 to 4	Nil	Nil	3 to 4	2 to 4	Nil
8	S. 5492	Nil	2 to 4	2 to 4	Nil	Nil	3 to 4	1 to 2	Nil
9	S. 8457	Nil	2 to 4	2 to 4	Oxalate	Nil	2 to 3	1 to 2	Nil
10	S. 9707	Nil	1 to 2	1 to 2	Oxalate	Nil	1 to 2	1 to 2	Nil
11	T. 101	Trace	2 to 4	2 to 4	Crystals	Nil	4 to 5	3 to 4	Nil
12	T.1201	Nil	2 to 4	2 to 4	Nil	Nil	2 to 4	2 to 4	Nil
13	T. 1362	+	2 to 4	2 to 4	Nil	Nil	1 to 2	1 to 2	Nil
14	T. 3053	Nil	2 to 3	1 to 2	Nil	Nil	1 to 2	1 to 2	Nil
15	T. 3220	Nil	1 to 2	1 to 2	Nil	Nil	2 to 3	2 to 3	Nil
16	T. 5894	Nil	2 to 4	5 to 6	Nil	Nil	2 to 4	2 to 4	Nil
17	T. 6924	Nil	3 to 4	1 to 2	Nil	Nil	2 to 3	1 to 4	Nil
18	T. 8061	Nil	2 to 4	2 to 4	Oxalate	Nil	2 to 3	1 to 2	Nil
19	T. 8379	Trace	1 to 2	1 to 2	Oxalate	Nil	2 to 4	3 to 4	Nil
20	T. 8456	Nil	3 to 4	2 to 4	Oxalate	Nil	4 to 6	4 to 6	Nil
21	T. 9592	Nil	2 to 4	1 to 2	Nil	Nil	1 to 2	1 to 2	Nil
22	U.1119	+	2 to 4	1 to 2	Oxalate	Nil	2 to 4	2 to 4	Nil
23	U.1656	Nil	3 to 3	1 to 2	Nil	Nil	3 to 4	3 to 4	Nil
24	U. 4075	Trace	5 to 6	2 to 4	Oxalate	Nil	3 to 4	2 to 3	Nil
25	U. 5278	Nil	3 to 4	1 to 2	Nil	Nil	4 to 6	4 to 6	Nil
26	U. 7537	Nil	2 to 4	2 to 4	Nil	Nil	2 to 3	2 to 3	Nil
27	U. 9107	Nil	3 to 4	1 to 2	Nil	Nil	2 to 3	2 to 3	Nil
28	V. 2027	Nil	2 to 3	2 to 3	Nil	Nil	1 to 2	1 to 2	Nil
29	V. 5386	Nil	2 to 4	3 to 4	Nil	Nil	2 to 3	2 to 3	Nil
30	V. 6251	Nil	2 to 3	1 to 2	Oxalate	Nil	2 to 4	2 to 4	Nil

Out of 30 patients of urine examination 6 patients had albumin, 11 patients had crystals and 2 patients had pus cells in urine before treatment. After treatment 100% patients were normal report.

ULTRASONOGRAPHY REPORTS IN BEFORE AND AFTER TREATMENT

S. No	O P No	Name	Age sex	Before treatment	After treatment
1	R. 8855	Mr. Kumar	41/M	3x3 mm and 4x4 mm Sized calculi seen in mid calyx of right kidney and left kidney	Normal report
2	S.511	Mr. Anandan	30/M	5mm and 7mm sized calculi seen in upper and lower pole of Rt. kidney. 7mm size calculus seen in upper pole of Lt. Kidney, 7mm size in Rt. V. U.J	3mm size calculus seen in mid pole of Rt. kidney. 2mm size calculus seen in upper pole of Lt. kidney. Stone removed in urine.
3	S. 2319	Mr. Rajan	29/M	Bilateral renal small calculi	Normal report
4	S.3568	Mr. Chandrasekar	45/M	4.4 mm and 4.7mm sized calculi seen in lower calyx and mid calyx of Lt. kidney with Calyceal system dilated.	Normal study
5	S. 4030	Mrs. Atchammal	54/F	A calculus 8mm size seen in Rt. V.U junction with pelvic Calyceal system and Ureter dilated.	Normal study
6	S. 5492	Mr. Dhanasekar	35/M	9mm size calculus seen in collecting system of Lt. kidney	4mm size calculus seen in Lt. kidney.
7	S.8457	Mr. Sathish Kumar	24/M	5mm and 5mm sized calculi seen in upper calyx of Rt. kidney and Lt. kidney	Normal study
8	S. 9707	Mr. Richard	50/M	5mm and 4mm sized calculi seen in mid calyx of Rt. kidney. 5mm size calculus seen in lower calyx of Lt. kidney.	4mm size calculus seen in mid calyx of Rt. kidney. 5mm size calculus seen in lower calyx of Lt. kidney.
9	T. 101	Mrs. Pattammal	60/F	Multiple calculi seen in mid pole of Rt. kidney, largest size 8mm. 1.3 mm size calculus seen in Rt. proximal Ureter junction with hydronehrosis.	Ureter stone moved to bladder.
10	T.1201	Mr. Brimanaidu	41/M	5mm size calculus seen in lower pole of Lt. kidney	3.2 sizes calculus seen in lower pole of Lt. kidney

11	T. 1362	Mr. Velraja	60/M	5mm size calculus seen in Rt. kidney	4mm size calculus seen in Rt. kidney
12	T.3053	Mr. Rashid	39/M	8mm size calculus seen in lower calyx of Rt. Kidney	Normal study
13	T.5894	Mrs. Epsi	58/F	A clump of calculi in size of 2.3 cm seen in pelvis of Rt. kidney	2.5 cm size calculi seen in Rt. kidney
14	T.8061	Mr. Unnikrishnan	35/M	Two calculi size of 5mm and 8mm are seen in Rt. mid calyx. 4mm size calculus seen in Rt. upper ureter. 10 mm size calculus seen in Rt. V. U junction with Hydronephrosis 7mm calculus seen in Rt. mid calyx.	Two calculi size of 5mm and 6mm seen in Rt. mid calyx. 4mm size calculus seen in Rt. upper calyx. 6mm size calculus seen in Lt mid calyx No Hydronephrosis, Ureter appears normal.
15	T.8379	Mr. Lourthu mary	60/F	Two calculi size of 8mm and 9mm seen in left lower calyx.	Normal study
16	T.8456	Mrs. Patchaiammal	56/F	8mm size calculus seen in Rt. kidney. 7 to 8mm size calculus seen in Lt kidney.	4mm and 5mm size calculi seen in Rt. and Lt kidney.
17	U. 7537	Mr. Sathesh kumar	25/M	5mm and 6mm size calculi seen in Rt. and Lt kidney	Normal study
18	U. 9107	Mrs. Mushina sheri	25/F	A calculus size of 5mm seen in Rt. mid calyx.	Normal study
19	V. 6251	Mr. Durai raj	23/M	1.5 cm size calculus seen in Lt lower calyx	9mm size calculus seen in Lt lower calyx

1	S. 359	Mr. Ranganathan	35/M	5mm size calculus seen in right mid calyx. Left Ureter and Calyceal system dilated	Stone removed In urine on 8/12/06
2	U. 5278	Mr. Raja	42/M	8.5 and 6mm size calculi seen in upper calyx of Rt. kidney. 5mm and 6mm size calculi seen in mid and lower calyx of Lt kidney. 10mm size calculus seen in Lt mid Ureter.	Stone removed in urine.
3	S.511	Mr. Anandan	30/M	5mm and 7mm sized calculi seen in upper and lower pole of Rt. kidney. 7mm size calculus seen in upper pole of Lf. Kidney, 7mm size in Rt. V. U.J	Stone removed in urine.
4	T. 101	Mrs. Pattammal	60/F	Multiple calculi seen in mid pole of Rt. kidney, largest size 8mm. 1.3 mm size calculus seen in Rt. proximal Ureter junction with hydronehrosis.	Stone removed in urine.

19 Patients are taken ultrasonography after treatment. Among 30 patients, in 9 patients calculi are totally reduced, in 9 patients calculi are reduced in size and number and one patient calculus not reduced.

Among 30 patients, ureter stone removed in urine in 4 patients.

9 patients are not taken U S G after treatment.

DISCUSSION

Urinary calculus is known to mankind since antiquity. The earliest recorded example being bladder and kidney stones detected in Egyptian mummies dated to 4800 B. C. **Kalladaippu** is most common disease of the present society. In spite of new approach in diagnosis and management like Percutaneous nephrolithotomy, spiral CT, extracorporeal shock-wave lithotripsy but it still continues to cause significant morbidity with high tendency of recurrence. **Kalladaippu** commonly affected in middle and low class family, they do not do these advance procedure.

The contribution of yoogi in the field of regional and humoral pathology regarding the disease **kalladaippu** is highly commendable. Siddha medicine is ancient duration system of medicine for centuries have provided proven treatment of disease like **kalladaipu**. There fore clinical trial was conducted on kalladaippu patients with **kalladaippu thool**, 30 patients of **kalladaippu** patients were selected.

1. DISTRIBUTION OF CASES BY GENTER

The gender distribution was 2.3: 1(M: F) in total of 30 patients, ie.21males and 9 females because increased serum testosterone causes increased endogenous oxalate production in male, increased urinary citrate in women.

2. DISTRIBUTION OF CASES BY AGE

A total of 30 patients of varying age group were included in this study, the maximum distribution of **kalladaippu** occurs evenly in the 31 to 40, 41 to 50 and 51to 60 years of age groups, 25 patients were in the age groups(e. g) 83.3% of the total number comes under the study. Several authors have reported 30 to 50 years of age group as the period of maximum incidence of **kalladaippu**, these periods are mainly in pittha period (33.4yr to 66.4yr) of human life as per the “Rathina surukka nadi”.

“வன்னநடு விற்பித்தமதா மப்பால்”
- கண்ணுசாமியம் லெண்டா

3. DISTRIBUTION OF CASES AMONG FOOD HABITS

According to table 4, 26 patients were on non vegetarian foods, these are induced vatham and pittham causes for **kalladaippu**. These food are having mainly protein and fat substances, it can induce humour of Vatham and pitham.

Animal protein increase urinary calcium, oxalates and uric acid and decrease urinary citrates excretion in urine due to protein contains sulphur contain amino acid, it increases endogenous acidosis.

Excessive in take of beverage likes coffee, tea, milk and alcohol are induces **kalladaippu** because of tea contain large amount of oxalates, coffee increase urinary calcium excretion, milk contains large amount of calcium and it has lactose potent stimulator of calcium absorption.

4. DISTRIBUTION OF CASES AMONG SEASON

While distribution of **kalladaippu** was highest in Elavenil, Muthuvenil and Mazhi kalaam, out of 30 cases 28 cases belong to these periods because of in Elavenil kalaam iyyam comes in normal, in Muthuvenil kalaam vatham increased and iyyam decreased, in Mazhi kalaam pitham increased above in normal level. Our literature mention as Vatham and pittham is one main cause for **kalladaippu**.

“வளிந்ததோர் வாதபித்தந் தால் வந்து
பெருங்கல்லாய் நீர்வழி யடைத்து”

Price and associates found that the incidence of urinary calculi was higher during the summer months. High temperatures increase perspiration, which may result in concentrated urine. This promotes increased urinary crystallization.

5. DISTRIBUTION OF CASES AMONG NILAM

Out of 30 cases 25 belonged to neithal nilam because in our literature reveals this land is a place of aggravation of Vatham and also the

food substances obtained here containing salt. Minerals contents of water may contribute to cause stone disease.

These environmental factors, diet factors and personal habits leads to derangement of Vatham and pitham, these humours are main predisposing factors for *kalladaippu*.

6. DISTRIBUTION OF CASES AMONG NEERKURI AND NEI KURI

On examination by envagai thervugal, the moothiram had been affected in all patients due to derangement of pitha kutram and abana vayu causing abdominal pain, yellowish urination, dysuria, Oliguria, nausea and vomiting.

7. DISTRIBUTION OF CASES AMONG CLINICAL FEATURES

As per the above data 100% of patients were affected abdominal pain and above 50% of patients were affected by yellowish urination, dysuria and Oliguria, after treatment abdomen pain relieved in 93.3% of patients, where other symptoms were relieved in 100% of patients.

The presence of albumin, crystals and pus cells come down to normal under treatment with trial drug in 100% of cases.

8. ULTRASONOGRAPHY REPORT

Out of 30 cases 19 patients were taken ultrasonography, after treatment with trial drug with in 8 weeks of periods, stone completely dissolved in 9 patients, ureter stone removed in urine in 4 patients and stone reduced in size and numbers in 9 patients. Other 9 patient's clinical symptoms were totally reduced.

9. BIOCHEMICAL STUDY

Potassium:

The trial drug having potassium salts, these are good diuretics and reduce urinary excretion of calcium and kidney stone recurrence.

Potassium citrate prevents upper urinary tract calculi this proved by Lee YH, HuangWc and Tsai JY in J. urology.1999 may: 161.

Citrates:

The drugs having Citrates is most important complexor of calcium in urine and reduce ionic calcium concentration, sodium citrate salts mixed with alkaline salt of trial drug causes metabolic alkalosis decrease mitochondrial uptake of citrate and increase urinary citrate excretion that prevention of recurrent calcium oxalate stones.

Magnesium:

Magnesium salts produce more favorable Magnesium to calcium ratio in urine in condition that offers relative protection against stone formation. Magnesium decrease renal tubular citrate resorption through the chelation of citrate and this increase urinary citrate excretion in urine. It salts lowered the relative saturation urine with calcium oxalate or brushite.

Phosphate:

Phosphates were used in the hope of binding dietary calcium in the intestinal thus reducing its absorption. Urinary phosphorus excretion is markedly increased during therapy with alkaline phosphate which results in an increasing in the inhibitor activity of calculus in urine perhaps because of increased renal excretion of pyrophosphate and citrate.

Zinc:

The potent inhibitory effect of urinary zinc excretion on urolithiasis evaluated in urologia internationalis 2004: 72:233-236.

10. PHARMACOLOGICAL STUDY:

Test drug ***kalladaippu thool*** (1000mg/kg/p.o) was given in water the diuretic activity was not significant at the end of 5 and 10 hrs when compared to urea (1000mg/kg/p.o).

However an enhanced diuresis was observed when test drug ***kalladaippu thool*** was along with radish juice with a Lipschits value more than 1 at the end of 5 and 10 hrs of examination.

11. ACUTE ORAL TOXICITY STUDY:

Kalladaippu thool at the dose of 2000 mg/kg/po did not exhibit any mortality in rats. As per the OECD 423 guideline the dose is said to be Unclassified under the toxicity scale.

12. TREATMENT:

All ingredients of trial drug are having diuretic and lithotriptic effects, they were prepared as per the formula seen in the Siddha literature.

13. A Patients were advised to take plenty of water and asked to follow strict diet restriction during and after treatment to prevent the recurrence of stones.

14. The clinical study was hope encouraging. This is only preliminary approach and study may be undertaken with the same drug in large number of patients, which may prove to be more effective in treatment.

SUMMARY

- The clinical trial was conducted in 30 patients of renal calculus with trial drug of ***kalladaippu thool*** in dose of 5gms twice a day with adjuvant of 50ml raddish juice.
- Clinical diagnosis of ***kalladaippu*** was done in the basis of clinical features described in our text book.
- Urine and haematological examination and ultrasonography were done before and after treatment of trial drug.
- The various Siddha aspect examinations were carried out and recoded.
- ***Kalladaippu*** in Siddha aspect was correlated with urinary calculus in modern aspect.
- In biochemical study the trial drug had magnesium, potassium, citrates and tannin. Which are confirmed the clinical prognosis in renal calculus.
- In pharmacological study showed that the trial drug had well diuretic action without adverse effect in dose of one gram per kg.
- The medicine chooses for clinical treatment and management of ***kalladaippu*** by ***kalladaippu thool***.
- Observation of the trial drug in the clinical study was clinically effective.

CONCLUSION

- The clinical trial of ***kalladaippu thool*** was conducted in 30 patients for 8 weeks of period.
- In the study 93.3 % of patients were clinically good improvement and 6.6% were moderate in improvement with trial drug. There were no clinically significant adverse reactions noted with trial drug.
- In Siddha and modern aspect of urine investigation in all patients after treatment came down to normal.
- Out of 30 patients, 19 patients were taken ultrasonography after treatment, among 19 patients, stone dissolved completely in 47.3 %(9) and reduced size and number in 47.3 %(9) of patients. Among 30 patients ureter stone removed in 13.3 %(4) with short period of treatment.
- In biochemical study the drug has potassium, magnesium, citrates and tannin were confirmed. This helps in clinical prognosis of renal calculus.
- The diuretic action of the trial drug had very good efficacy with anubanam in animal study and without toxic effect.
- Expenditure of the trial drug is low in cost and easily preparable but highly efficacy in ***kalladaippu***.
- The clinical study was encouraging to do the deep study in large sample of kalladaippu patients with trial drug with proper diagnosis.

PREPARATION AND PROPERTIES OF TRIAL DRUG

KALLADAIPPUHOL (கல்லடைப்புத் தூள்)

INGREDIENTS

1. நாயுருவி (Ash of Achyranthus aspera) -2 palam (70gms)
2. வாழை (Ash of Plantain barks) -2 palam (70gms)
3. பனை பூ (Ash of Palm flowering stalks) -2 palam (70gms)
4. கத்திரி (Ash of dried Brinjal's plants) -2 palam (70gms)
5. கத்தக்காம்பு (Powder of Pale catechu) -2 palam (70gms)
6. வெங்காரம் (Purified Borax powder) -2 palam (70gms)
7. படிகாரம் (Purified Alumen) -2 palam (70gms)
8. இந்துப்பு (Purified Rock salt) -2 palam(70gms)
9. சவுக்காரம் (Purified Sodium carbonates) -2 palam(70gms)

METHOD OF PREPARATION

Above all ingredients were taken in to equally and made powder form by rock mortar.

DOSAGE: 5gms (1 to 1.5 varagan eadai), twice a day for 8 weeks.

ADJUVANT: 50ml of Raddish juice

MEDICINAL USES: Urinary calculus, anuria, burning micturition, urethral stricture and tumor in urinary tract.

- அனுபாகவைத்திய நவநீதம், ஒன்பதாம் பாகம், பக்கம் 76.

-ஹக்கிம் பா. முகம்மது அப்துல்லா சாயபு

1.நாயுருவி (ACHYRANTHUS ASPERA)

Family: Amaranthaceae

Part of uses: Whole plants

Taste: Bitter, pungent and astringent,

Potency: Heat

Pirivu: Pungent

Action: Astringent, diuretic and alterative

பொதுக்குணம் (Therapeutic effects)

“மலிகாரங் கைப்புள்ள அபமார்க்கி யின்வேரால் வசிய முண்டாம்
இலைமூல உதிரமந்தம் பேதிகபம் வியர்வுதந்தி யிறங்கு மேகம்
மலையேறும் படிபுரியு முள்ளரிசி பசுமாற்றும் வசன மூலம்
பலமாதர்க் குள்ளுக்கை நீக்குவங்கச் சிந்துரம் பண்ணுமாதோ”
- (தே.கு)

Medicinal uses:

Decoction of whole plants is good diuretics in renal calculus.

It contains a large percentage of alkaline ash and it mainly contains potash salt. Decoction of whole plants is a good diuretic found efficacious in renal dropsies and general anasarca.

Ashes of plants with water, jaggery cures dropsies such as anasarca, ascites.

-Indian materia medica, by.Dr.K.M.Nadkarni.

It is useful in renal and vesical calculi, flatulence, colic, painful inflammation, dropsy, and vomiting.

- Indian medicinal plants, Volume-1. Vaidyaratnam, P.S.Varier's.

2. வாழை (MUSA PARADISIACA)

Family: Mimosaceae

Part of uses: Fruit, leaves and stems

Taste: Astringent

Potency: Cold

Pirivu: Pungent

Action: Styptic, anti Vatha, diuretic, refrigerant and astringent.

பொதுக்குணம் (Therapeutic effects)

“வாழைநீர் தான்குளிர்ச்சி வல்லபலமுண்டாகும்
பேழை வயிறுடைக்கும் பெண்மயிலே வீழுவல்லி
ரத்தக் கிரிச்ச மெரிநீரிவையுடனே
சிற்றிரணம் போக்குந்தெறி” - (அ.கு)

Medicinal uses:

It cures ascites, burning micturition, haematuria and abdominal ulcer. Its salt is a diuretic and lithotriptic action, it prepared from plantain bark ashes. Plantain ash contains phosphoric anhydride, lime, alkalies, iron, and chlorine etc.

Ash of husk of ripe fruit contains carbonates of potash and soda, chloride of potassium, alkaline phosphates with a little sulphate, lime, silica, earthy phosphates etc. Ashes produced by burning the plant contain potash salt and are therefore useful in acidity, heartburn and colic.

Its ashes is a potash salt uses for nephritis, uraemia and ascities.

-Indian materia medica By Dr.K.M.Nadkarni.

The ashes obtained by burning plant are anti scorbutic, anthelmintic, and stomachic, and are useful in hyperacidity, heartburning, colic, and verminosis. The inflorescence axis (stem) is very specific for renal and vesical calculi.

-Indian medicinal plants, Vaidyaratnam, P.S.Varier.

3. பனை பூ (BORASSES FLABELLIFER)

Family: Palmaecae

Part of uses: Root, flowering stalk, juice, bark and fruits.

Taste: Astringent

Potency: Cold

Pirivu: Sweet

Action: Astringent, diuretic, demulcent, nutritive and refrigerant.

பொதுக்குணம் (Therapeutic effects)

“பனையிலுறு பூலதுதான் கங்கமுறாக் குன்ம
வினையகற்றும் நீர்கட்டை மீட்டும் முனையான
பன்னோய் ஒழிக்கும் பழஞ்சுரத்தைப் போக்கிவிடும்
முன்னே இதனை விளம்பு” - (அ.கு)

Medicinal uses:

It cures vayu gunmam, burning micturition, dental disease and chronic fever. Ashes of flowering stalk are diuretics. Salt prepared from ashes of flowering stalk diluted with water then filter it pure ash less diluted water exposure to sun light. It contains albuminoids gum and fat. Juice is diuretics, cooling, stimulant, and antiphlogestic.

It uses for dropsy and inflammatory condition. Ashes of flavoring stalk are useful in enlarged spleen.

- Indian materia medica
By.Dr. K.M.Nadkarni.

The ash obtained by burning the inflorescence is good antacid, antiperiodic, and useful in hyperacidity, heartburn, biliousfever, splenomegaly, hepatomegoly, and skin diseases.

- Indian medicinal plants, volume-1
Vaidyaratnam, P.S.Varier's.

4. கத்திரி (SOLANUM MELONGENA)

Family: Solanaceae

Part of uses: All parts of plant

Taste: Bitter and astringent

Potency: Heat

Pirivu: Pungent

Action: Stimulant, hypnotic and expectorant.

பொதுக்குணம் (Therapeutic effects)

“கத்திரிக்காய் பித்தங் கனன்றகபந் தீர்த்துவிடும்
தொத்த சொறிசிரங்கைத் தூண்டிவிடும் மெத்தவந்தான்
பிஞ்சான கத்திரிக்காய் பேசுமுத்தோ டம்போக்கும்
மஞ்சார் குழலே வழுத்து” - (அ.கு)

Chemical constituents:

Dried material contain ether extract 4.2 p.c., albuminoids 16.37p.c., nitrogen 2.62p.c., soluble carbohydrate 55.23 p.c., woody fibre 17pc ash 7.20p.c.

Ripe ones are carbonaceous and bilias. Burnt fruits are like in digestion, purgative, slightly cilious and beneficial in phlegm, wind and obesity.

-Indian materia medica

Medicinal uses: It leaves used in diuretic drugs.

5. கத்தக்காம்பு (UNCARIA CAMBIR)

Family: Rubiaceae

Part of uses: Stem

Taste: Bitter astringent

Potency: Heat

Pirivu: Pungent

Action: Astringent.

பொதுக்குணம் (Therapeutic effects)

“பேதிசிறு நீர்கட்டும் பேசுவொணா வாய் விரணமும்
தீதிலுய ருந்திப்புண் தீருமே மேதினியில்
செங்குருதி தோசமெலாம் தீர்ந்துவிடும் விந்திறுகும்
பைன்கூந்தல் மாமே பகார்’

-சித்த வைத்திய பதார்த்த குண விளக்கம்,
சி. கண்ணுசாமிப்பிள்ளை.

Medicinal uses:

It cures diarrhea, anuria, oral ulcer, burned ulcer, blood diseases and increase concentration of semen.

It contains the active principle- Catechu- tannic acid 22 to 50%p.c, catechine 7 to 33 p.c., quercetin, a yellow – colouring principle, catechu-red, gambier- fluorescein, wax and oil etc.

-Indian materia medica by. Dr.K.M.Nadkarni

5. வெங்காரம் (SODIUM BIBORATE)

It is composed of boric acid and soda.

Taste: Sweet with astringent.

Potency: Heat

Pirivu: Pungent

Action: Astringent, diuretic, antacid, local sedative, antiseptic and emmenagogue.

பொதுக்குணம் (Therapeutic effects)

“சொறிபுடை யெண் குன்மநமை சோரியாசம்
பறிகிரகணி கல்லூனம் பன்னோய் நெறியைத்
தடங்கணங்க பங்கிருமி சர்ப்பவிடஞ் சந்நி
யிடங்கணங்க லக்கிற்போ மெண்”

-Siddha materia medica

Purification:

It fried by heat, still water contents removal.

Medicinal uses:

It cures scabies, eight types of gunmam, giddiness, dysentery, urinary calculus, burning micturition, dental diseases, worm's infection, snake poison and epilepsy.

Vengara parpam cures pitha diseases like burning micturition and kalladaippu.

Borax is given orally in dose varying from 10 to 30 grains in acidity of stomach, dyspepsia and intestinal organism. It commonly mixed in decoction for kalladaippu.

6. படிக்காரம் (ALUMEN)

Characters:

It is colorless, transparent crystals, with acid. Alum is a general name for a class of double sulphates containing aluminium and such metals as potassium, ammonium, iron, etc.

Taste: Sweetish astringent

Potency: Heat

Pirivu: Pungent

Action: Astringent, diuretic, haemostatic and anti spasmodic.

பொதுக்குணம் (Therapeutic effects)

“சீனமெனும் காரமது சீறிவரு பல்லரணை
ஆனைக்கால் கண்ணோய் அனிலமொடு மாநிலத்தில்
துன்மாங்கிசம் வாயு தோலாத உள்ளழலை
குன்மமலை போக்குமெனக் கூறு”

-Siddha materia medica

Purification:

It makes in to power form and mixed with water then boiled well up to solid consistence and tried with sun light.

Medicinal uses:

It cures gingivitis, fileriasis, eye diseases, heart diseases, Vatham, fever, peptic ulcer and tumor. Padikara parpam has been used for urinary calculus, urethral stricture, anuria and burning micturition.

–Siddha materia medica.

It is useful in leucorrhoea, haematuria, haemoptysis, menorrhagia, gastric and intestinal catarrh and other haemorrhages.

-Indian materia medica, by.Dr.K.M.Nadkarni.

7. இந்துப்பு (SODIUM CHLORIDE IMPURA)

Character:

It is found in small white crystalline grains or transparent cubes. It is brownish white externally and white internally. It has a pure saline taste and burns with a yellow flame Taste: Salt in taste.

Potency: Heat

Pirivu: Pungent,

Action: Diuretic, laxative, carminative and stomachic.

பொதுக்குணம் (Therapeutic effects)

“அட்ட குன்ம மந்தம் அசிர்க்கரஞ்சூர் சீதபித்தந்
துட்டவையம் நாடிப்புண் டோடங்கள் கெட்டமலக்
கட்டுவிட விந்தையக் காமியநோய் வன்கரப்பான்
விட்டுவிட விந்துப்பை விள்’

-Siddha materia medica

Purification:

It is made into powder form, mixed with anna kaadineer, kept in to the sun light for three Days, then washed with fresh water and dried with sun light.

Medicinal uses:

It cures eight types of gunmam, dyspepsia, kaba pitham, tumour, three thosam, constipation, poison, sukila noi and eczema.

In small dose it is highly carminative, stomachic and digestive. It possesses stronger purgative property. -Indian materia medica

By.Dr. K.M.Nadkarni.

8. சவுக்காரம் (SODIUM CARBONATE)

“தீய சவுக்காரஞ் செப்புவேன் கேளு
மாயாதி பூனீர் வார் வெடிச்சாரமுந்
தோய்வெடிநீரிற் சுண்டிடக் காய்ச்சி
வாயூர யெண்ணை வடித்து உருட்டிடே
உருட்டிய காரம் ஓம் சவுக்காரமும்
மருட்டிடுஞ் சரக்கை வாட்டி யெடுத்திடு
திருட்டுகள் பேசி திரிகிற வாதியை
மெருட்டு மிக்காரம் வேதைகள் கோடியே”
மச்சமுனி 800 பக்கம் 24

Medicinal uses sowkaram was prepared by fuller earth, pooneeru, potassium nitrates and lime.

பொதுக்குணம்: (Therapeutic effects)

“கார்மேகத் தூர்ப்படையும் கட்டிக் கரப்பனுடன்
கார்மேக வூற்ற் றமுபலரும் சேர்மேகத்
தொட்டி வரும் நகத்தினூற வகற்றிவிடும்
கட்டிச் சவுக்காரங் காண்”

-சித்த வைத்திய பதார்த்த குண விளக்கம்,
சி. கண்ணுசாமிப்பிள்ளை.

Fuller earth is one of the ingredients of sowkaram so it has same action.

Constituents: Fuller earth contains 25 p.c. of sodium carbonate with certain impurities such as organic matter, sulphate of soda, potash.

Action: It is antacid, alterative, and diuretic.

Medicinal uses:

It is useful in dyspepsia with vomiting, diarrhea, and flatulense. It is efficient remedy in urinary diseases as uric acid, gravel and suppression of urine.

-Indian material medica by.Dr.K.M.Nadkarni.

ADJUVANT:**RADDISH JUICE (RAPHANUS SATIVAS, Linn):**

Family: Cruciferae

Part of uses: All parts of plant

Taste: Pungent

Potency: Cold

Pirivu: Pungent

Action: Diuretic, Laxative. Stimulant, Stomach and Aphrodisiac

பொதுக்குணம்: **(Therapeutic effects)**

“வாதங் கரப்பான் வயிற்றெரிவு சூலைகுடல்
வாதங்கா சமையும் வன்தலை நோய் மோதுநீர்க்
கோவைபன்னோய் பல்சிலந்தி குன்மமிரைப் புக்கடுப்புஞ்
சாவுமிள்ளங் கிக்கந்தத் தால்”

-Siddha materia medica

Constituents:

Fresh vegetable contain 91.00 p.c of moisture.

Completely dried material contain etherextract-4.00p.c,

Albuminoids-18.00p.c, Solublecarbohydrates-52.66p.c,

Woody fibre- 9.34 p.c, and Ash-16.00 p.c.

Seeds and wood contain fixed oil and sulphureted volatile oil

Medicinal uses:

Root juice given for urinary complaints like dysurea and strangury.

-Indian materia medica

By.Dr. K.M.Nadkarni.

Root juice 34ml to 100ml is increased urination.

Root is contains rich in vitamin C and E it increase immunological and antibacterial activity of white blood cells by increasing their motility.

**QUALITATIVE ANALYSIS OF ACIDIC/BASIC RADICALS AND
PHYTOCHEMICAL CONSTITUENTS IN TEST DRUGS**

Procedure	Observation	inference
Test for Calcium: 2 ml of extract is taken in a clean test tube. To this add 2 ml of 4% ammonium oxide solution.	White precipitate is formed	Presence of calcium
Test for Sulphate : 2 ml of the extract is added to 5 % barium chloride solution.	White precipitate is formed	Presence of Sulphate
Test for Chloride : The extract is treated with Silver nitrate solution	White precipitate is formed	Presence of Chloride
Test for carbonate : The substance is treated with Conc. HCl.	Effervescence is formed	Presence of carbonate
Test for Starch : The extract is added with weak iodine solution	Blue colour is not formed	Absence of starch
Test for Iron (Ferric) : The extract is treated with glacial acetic acid and potassium ferrocyanide	Blue colour is formed	Presence of Ferric iron
Test for Iron (Ferrous) : The extract is treated with Conc. HNO_3 and ammonium thiocyanate	No Blood red colour is formed	Absence of Ferrous iron
Test for phosphate : The extract is treated with	Yellow precipitate is	Presence of phosphate

ammonium molybdate and conc. HNO_3	formed	
Test for Tannic acid : The extract is treated with Ferric chloride	Blue black precipitate is formed	Presence of Tannic acid
Test for Unsaturation : 1 ml of Potassium permanganate solution is added to the extract.	Does not get decolourised	Absence of unsaturated compound
Test for saponins: Dilute extract + 1ml of distilled water and shake well.	Froth formation	Presence of saponins
Test for sugars : Benedict method ; 5ml of Benedict solution heated gently then add 8 drops of diluted extract then heated in a boiling water bath. Molisch test; Dilute extract + 2 drops of Molisch+3ml conc. H_2SO_4 .	No colour change No Reddish violet zones appeared	Indicates the Absence of sugar Absence of carbohydrate
Test for steroids : Liberman Burchard test; Dilute extract + 2 ml acetic anhydride + conc. H_2SO_4 .	No Formation of red colour	Absence of steroids
Test for amino acids: Dilute extract + 2ml of Ninhydrin's soln.	Formation of violet colour	Presence of amino acids
Test for proteins: Biuret method ; 1ml of dilute	Formation of Violet	Presence of proteins

extract + 1ml of 5% CuSO ₄ + 1% NaOH.	colour	
Test for Flavanoids : Dilute extract + 2 drops of conc. HCl and gently heated.	No formation of pink colour	Absence of Flavanoids
Test for phenol; Dilute extract + 2 drops of FeCl ₃ soln.	Deep green colour is formed	Presence of phenols
Test for Tannins; Dilute extract + 2 ml of 10% lead acetate adds.	White precipitate formed	Presence of tannins
Test for alkaloids; Mayer's method; 1 ml of dilute extract + 1 ml reagent. Dragendorff's method; 1 ml of dilute extract + 1 ml of reagent.	No appearance of cream colour precipitate No appearance of orange colour precipitate	Absence of alkaloids Absence of alkaloids
Citrate: 2 ml dilute extract add 0.5 ml of sulphuric acid, and 3 ml of KMnO ₄ warm until the colour of KMnO ₄ is discharged and add 10% of sodium nitro prusside in 1M of sulphamic acid make in to alkaline with strong ammonia	Violet colour turns in to violet in blue.	Presence of citrate

solution.		
Magnesium: 2ml dilute extract of sample + 1ml of diluted ammonia	White precipitate formed	Presence of magnesium
Ammonia: 2ml dilute extract of sample add with 2M NaOH and 0.3ml of potassium tetra iodomercurate solution and allowed stand for 5 minutes.	Yellow colour developed	Presence of ammonia
Zinc: 2ml dilute extract of sample add with 1.5 ml of 2 M HCl, 5ml of distilled water and diluted sodium hydroxide.	White precipitate formed	Presence of Zinc

PRECLINICAL PHARMACOLOGICAL AND TOXICOLOGICAL STUDY OF KALLADAIPPU THOOL FOR DIURETIC ACTIVITY IN RATS

Index

1.0 Materials and Methods

- 1.1 Test drugs**
- 1.2 Preparation of drugs for dosing**
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2.0 Results

- 2.1 Preliminary phytochemical screening**
- 2.2 Acute oral toxicity study**
- 2.3 Diuretic activity**

3.0 Discussion

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1.0 MATERIALS AND METHODS

1.1 Test Drugs

The test drug used in the study was processed by the methods prescribed in standard text books of siddha medicines.

1.1 Kalladaippu thool

Kalladaippu thool was prepared by the method described in Anuboga vaidhya navaneetham 9th part, page no.72.

1.2 Preparation of drug for dosing

Drug used for the study was suspended each time with 1% (w/v) solution of sodium carboxy methyl cellulose before administration.

1.3 Drugs and chemicals

Fine chemicals used in these experiments were obtained from Sigma Chemicals Company, U.S.A. Other analytical grade chemicals were obtained from S.d. Fine Chemicals Ltd., Mumbai.

1.4 Experimental animals

Colony inbred wistar rats of either sex weighing 200 - 250 g were used for the pharmacological and toxicological studies. The animals were kept under standard conditions 12:12 (day/night cycles) at 22⁰C room temperature, in polypropylene cages. The animals were fed on standard pelleted diet (Hindustan Lever Pvt Ltd., Bangalore) and tap water *ad libitum*. The animals were housed for one week in polypropylene cages prior to the experiments to acclimatize to laboratory conditions. The experimental protocol was approved by the Institutional Animal Ethical Committee (IAEC).

1.5 Acute oral toxicity study

Acute oral toxicity was conducted as per the OECD guidelines (Organization of Economic Cooperation and Development) 423 (Acute Toxic Class Method). The acute toxic class method is a stepwise procedure with 3 animals of a single sex per step. Depending on the mortality and /or moribund status of the animals, on the average 2-4 steps may be necessary to allow judgment on the acute toxicity of the test substance. This procedure results in the use of a minimal number of animals while allowing for acceptable data based scientific conclusion.

The method uses defined doses (5, 50, 300, 2000 mg/kg body weight) and the results allow a substance to be ranked and classified according to the Globally Harmonized System (GHS) for the classification of chemicals which cause acute toxicity

Wistar albino rats of either sex weighing 200-250 g were fasted overnight, but allowed water *ad libitum*. Since the formulation is relatively non toxic in clinical practice the highest dose of 2000 mg/kg/p.o (as per OECD guidelines “Unclassified”) was used in the acute toxicity study.

The animals were observed closely for behavioural toxicity, if any by using FOB (Functional observation battery).

1.6 Diuretic activity in rats (Lipschitz test)

A method for testing diuretic activity in rats has been described by Lipschitz *et al.*, 1943. The test is based on urine excretion in test animals and compared to rats treated with a high dose of urea. The “Lipschitz value” is the quotient between urine excretion by test animals and urine excretion by the urea control.

Male wistar rats weighing 150-200 g were used. Three animals per group are placed in metabolic cages provided with SS wire mesh bottom and funnel to collect the urine. SS sieves are placed on the funnel to retain

feces and allow the urine to pass. The animals were fed with standard pelleted diet and water *ad libitum*. 15 hrs prior to the experiment food and water were withdrawn. The animals were divided into 4 groups with 3 animals in each group and the following regimen of treatment was instituted.

Group I	Control (Radish juice) 5 ml/per animal
Group II	Urea (Standard) 1g/kg/p.o
Group III	Test drug in water 1000 mg/kg/p.o
Group IV	Test drug (1000 mg/kg/p.o) in radish juice (5ml/per animal)

For screening procedures 2 groups of three animals were used for the study. The test drug ***Kalladaippu thool*** and standard drug urea were administered orally at the doses of 1g/kg, respectively. Additionally 5 ml of 0.9% sodium chloride per 100 g body weight was given by gavage. Urine excretion was recorded after 5 and 10 hrs.

Urine volume excreted per 100 g body weight was calculated for each group. Results are expressed as “Lipschitz value” ie, the ratio of test/urea. Indices of 1.0 and more are regarded as a positive effect.

2.0 Results

2.1 Preliminary basic, acidic radicals and phytochemical studies

The qualitative chemical analysis and acidic, basic radical's assay of the drugs showed the presence of phytoconstituents and minerals as depicted in (Table 1).

2.2 Acute oral toxicity study

Kalladaippu thool at the dose of 2000mg/kg/po did not exhibit any mortality in rats. As per OECD 423 guidelines the dose is said to be "Unclassified" under the toxicity scale. Hence further study with higher doses was not executed.

2.3 Diuretic study

In the diuretic study there was no significant diuretic activity was observed when radish juice alone is given at the end of 5 and 10 hrs observation. Where as urea (1 g/kg/p.o) showed significant diuretic activity at the end of 5 and 10 hrs of observation. When the test drug ***Kalladaippu thool*** (1000 mg/kg/p.o) was given in water the diuretic activity was not significant at the end of 5 and 10 hrs when compared to standard urea (1g/kg/p.o). However an enhanced diuresis was observed when the test drug ***Kalladaippu thool*** was given along with radish juice with a Lipschitz value more than 1 at the end of 5 and 10 hrs of observation. This study clearly establishes the enhanced diuretic activity of ***Kalladaippu thool*** when it was given with radish juice as "anupanam". The animals received radish juice alone (5 ml/per animal) did not exhibit significant diuretic activity.

Discussion

The ***Kalladaippu thool*** is said to be clinically useful siddha formulation for diuresis. In the present study the acidic and basic radicals and phytochemical studies revealed the presence of K^+ , Na^+ , Ca^{++} , Fe^{+++} , Mg^{++} , Zn^{++} , Sulphate, Chloride, Carbonate, Phosphate, Citrate, Ammonium radicals and phytoconstituents like tannins, tannic acid, phenols etc.

In clinical practice the drug is given at the divided doses of 10 g/day in radish juice. The radish juice is used as “anupanam” for enhancing the absorption and bioavailability of the test drug. Administration of radish juice alone did not exhibit any significant diuretic activity but when co administered with ***Kalladaippu thool*** exhibited significant diuretic activity. Similarly ***Kalladaippu thool*** administered in water did not exhibit significant diuretic activity when compared to standard drug urea.

The “Lipschilz value” of present study shows a π/U ratio of more than one (1) which is considered to be a significant index to rate a drug as diuretic.

There is good correlation in diuretic activity of ***Kalladaippu thool*** in both clinical and experimental study when co-administered with radish juice. The presence of Na^+ and K^+ ions and other basic and acidic radicals present in the formulation may be responsible for the diuretic activity of ***Kalladaippu thool***. The exact mechanism of diuretic action of ***Kalladaippu thool*** should be investigated further.

Table
Diuretic activity of *Kalladaippu thool* in rats

Groups	5 hrs	10 hrs
Control (Radish)	0.7167±0.2927	2.017±0.4070 ^a
Urea (std)	1.33±0.1751 ^a	3.333±0.3882 ^{a,c}
Test drug (water)	1.23±0.28 ^{a,b}	1.367±0.1633 ^a
Test drug (radish)	1.733±0.2066 ^{a,d,e}	3.550±0.2811 ^{a,d,e}

n = 6, values are expressed as mean ± S.D followed by One way Anova using Turkey's multiple comparison method.

P<0.001 was considered significant.

ap< 0.001, bp< 0.01, cp<0.001, dp>0.05, ep<0.001

- a - all groups vs control
- b,c - test (water) vs standard
- d - test (radish) vs standard
- e - test (water) vs test (radish)

**NATIONAL INSTITUTE OF SIDDHA,
CHENNAI 47**

**AN OPEN TRIAL OF *KALLADAIPPU THOOL* FOR THE
TREATMENT OF *KALLADAIPPU* (URINARY CALCULUS)**

FORM-I SELECTION PROFORMA

1. O.P.No / I.P No: _____ 2. Bed No: _____ 3. S.No: _____

4. Name: _____ 5. Age (years): ____ 6. Gender: M ☐ F ☐

7. Occupation: _____

8. Address: _____

9. **COMPLAINTS AND DURATION:** _____

10. **HISTORY OF PRESENT ILLNESS:** _____

PAST HISTORY

	1. Yes	2. No
11. Renal tubular obstruction:	<input type="checkbox"/>	<input type="checkbox"/>
12. Recurrent renal calculus:	<input type="checkbox"/>	<input type="checkbox"/>
13. Prolonged immobilization:	<input type="checkbox"/>	<input type="checkbox"/>
14. Kidney transplantation:	<input type="checkbox"/>	<input type="checkbox"/>
15. Healed renal tuberculosis:	<input type="checkbox"/>	<input type="checkbox"/>

ENDOCRINE DISORDERS

- | | 1. Yes | 2. No |
|---------------------------|--------------------------|--------------------------|
| 16. Hyper parathyroidism: | <input type="checkbox"/> | <input type="checkbox"/> |
| 17. Cushing syndrome: | <input type="checkbox"/> | <input type="checkbox"/> |

CONGENITAL DISORDER HISTORY

- | | 1. Yes | 2. No |
|-----------------------------------|--------------------------|--------------------------|
| 18. Medullary sponge kidney: | <input type="checkbox"/> | <input type="checkbox"/> |
| 19. Myelo proliferative disorder: | <input type="checkbox"/> | <input type="checkbox"/> |

TREATMENT HISTORY

- | | 1. Yes | 2. No |
|---|--------------------------|--------------------------|
| 20. Oral diuretics: | <input type="checkbox"/> | <input type="checkbox"/> |
| 21. Non- steroid anti- inflammatory drugs: | <input type="checkbox"/> | <input type="checkbox"/> |
| 22. Sugery for urinary calculus: | <input type="checkbox"/> | <input type="checkbox"/> |
| 23. Extra carporeal shock wave lithotripsy: | <input type="checkbox"/> | <input type="checkbox"/> |

FAMILY HISTORY

- | | 1. Yes | 2. No |
|------------------|--------------------------|--------------------------|
| 24. Gout: | <input type="checkbox"/> | <input type="checkbox"/> |
| 25. Cystinuria: | <input type="checkbox"/> | <input type="checkbox"/> |
| 26. Xanthinuria: | <input type="checkbox"/> | <input type="checkbox"/> |

DIET HISTORY

Vegetarian foods

- | | 1. No | 2. Yes |
|------------------------------------|--------------------------|--------------------------|
| 27. High intake of dairy products: | <input type="checkbox"/> | <input type="checkbox"/> |
| 28. Low calcium diet: | <input type="checkbox"/> | <input type="checkbox"/> |
| 29. Nuts | <input type="checkbox"/> | <input type="checkbox"/> |

Non- vegetarian foods

- | | | |
|--------------|--------------------------|--------------------------|
| 30. Meat: | <input type="checkbox"/> | <input type="checkbox"/> |
| 31. Poultry: | <input type="checkbox"/> | <input type="checkbox"/> |
| 32. Fish: | <input type="checkbox"/> | <input type="checkbox"/> |

Drinks

- | | | |
|--------------|--------------------------|--------------------------|
| 33. Alcohol: | <input type="checkbox"/> | <input type="checkbox"/> |
| 34. Tea: | <input type="checkbox"/> | <input type="checkbox"/> |
| 35. Coffee: | <input type="checkbox"/> | <input type="checkbox"/> |
| 35. Milk: | <input type="checkbox"/> | <input type="checkbox"/> |

GENERAL EXAMINATION

- | | |
|--------------------------------|---|
| 37. Weight (kg): | <input type="text"/> <input type="text"/> <input type="text"/> |
| 38. Temperature (°F): | <input type="text"/> <input type="text"/> <input type="text"/> . <input type="text"/> |
| 39. Pulse rate / minute: | <input type="text"/> <input type="text"/> <input type="text"/> |
| 40. Heart rate / minute: | <input type="text"/> <input type="text"/> <input type="text"/> |
| 41. Respiratory rate / minute: | <input type="text"/> <input type="text"/> <input type="text"/> |
| 42. Blood pressure (mmHg): | <input type="text"/> <input type="text"/> <input type="text"/> / <input type="text"/> <input type="text"/> <input type="text"/> |

- | | 1. Yes | 2. No |
|-----------------------------|--------------------------|--------------------------|
| 43. Anaemia: | <input type="checkbox"/> | <input type="checkbox"/> |
| 44. Jaundice: | <input type="checkbox"/> | <input type="checkbox"/> |
| 45. Cyanosis: | <input type="checkbox"/> | <input type="checkbox"/> |
| 46. Lymphadenopathy: | <input type="checkbox"/> | <input type="checkbox"/> |
| 47. Pedal oedema: | <input type="checkbox"/> | <input type="checkbox"/> |
| 48. Clubbing: | <input type="checkbox"/> | <input type="checkbox"/> |
| 49. Jugular vein pulsation: | <input type="checkbox"/> | <input type="checkbox"/> |

VITAL ORGANS EXAMINATION:

- | | 1. Normal | 2. Affected |
|-----------|--------------------------|--------------------------------|
| 50. Heart | <input type="checkbox"/> | <input type="checkbox"/> _____ |
| 51. Lungs | <input type="checkbox"/> | <input type="checkbox"/> _____ |
| 52. Brain | <input type="checkbox"/> | <input type="checkbox"/> _____ |

53. Liver	<input type="checkbox"/>	<input type="checkbox"/>	_____
54. Kidney	<input type="checkbox"/>	<input type="checkbox"/>	_____
55. Spleen	<input type="checkbox"/>	<input type="checkbox"/>	_____
56. Stomach	<input type="checkbox"/>	<input type="checkbox"/>	_____
57. Urinary bladder	<input type="checkbox"/>	<input type="checkbox"/>	_____

CLINICAL EXAMINATION

SIGNS AND SYMPTOMS

58. Abdominal pain	<input type="checkbox"/>	1. No <input type="checkbox"/>	2. Yes <input type="checkbox"/>
59. Severity:	0. NA <input type="checkbox"/>	1. Mild <input type="checkbox"/>	2. Moderate <input type="checkbox"/>
	3. Severe <input type="checkbox"/>		
60. Sites:	0. NA <input type="checkbox"/>	1. Loin <input type="checkbox"/>	2. Renal angle <input type="checkbox"/>
	3. Lower abdomen <input type="checkbox"/>		
61. Radiating areas:	0. NA <input type="checkbox"/>	1. Groin <input type="checkbox"/>	
	2. Thigh <input type="checkbox"/>	3. External Genitalia <input type="checkbox"/>	
		1. Yes	2.No
62. Abdominal distention	<input type="checkbox"/>	<input type="checkbox"/>	
63. Dysuria	<input type="checkbox"/>	<input type="checkbox"/>	
64. Oliguria	<input type="checkbox"/>	<input type="checkbox"/>	
65. Yellowish urination	<input type="checkbox"/>	<input type="checkbox"/>	
66. Haematuria	<input type="checkbox"/>	<input type="checkbox"/>	
67. Pyuria	<input type="checkbox"/>	<input type="checkbox"/>	
68. Nausea	<input type="checkbox"/>	<input type="checkbox"/>	
69. Vomiting	<input type="checkbox"/>	<input type="checkbox"/>	
70. Fever	<input type="checkbox"/>	<input type="checkbox"/>	
71. Sweating	<input type="checkbox"/>	<input type="checkbox"/>	

SIDDHA SYSTEM OF EXAMINATION

IYMPORI

	1. Normal	2. Affected
72. Mei	<input type="checkbox"/>	<input type="checkbox"/> _____
73. Vaai	<input type="checkbox"/>	<input type="checkbox"/> _____
74. Kan	<input type="checkbox"/>	<input type="checkbox"/> _____
75. Mookku	<input type="checkbox"/>	<input type="checkbox"/> _____
76. Sevi	<input type="checkbox"/>	<input type="checkbox"/> _____

KANMENTHIRIUM

77. Kai	<input type="checkbox"/>	<input type="checkbox"/> _____
78. Kaal	<input type="checkbox"/>	<input type="checkbox"/> _____
79. Vaai	<input type="checkbox"/>	<input type="checkbox"/> _____
80. Eruvai	<input type="checkbox"/>	<input type="checkbox"/> _____
81. Karuvaai	<input type="checkbox"/>	<input type="checkbox"/> _____

82. PARUVA KAALA

- | | |
|--|--|
| 1. Elavenil kalam <input type="checkbox"/> | 2. Muthuvenil kalam <input type="checkbox"/> |
| 3. Mazhai kalam <input type="checkbox"/> | 4. Kulir kalam <input type="checkbox"/> |
| 5. Munpani kalam <input type="checkbox"/> | 6. Pinpani kalam <input type="checkbox"/> |

83. THINAI

- | | | |
|-------------------------------------|------------------------------------|--------------------------------------|
| 1. Kurunji <input type="checkbox"/> | 2. Mullai <input type="checkbox"/> | 3. Marutham <input type="checkbox"/> |
| 4. Neithal <input type="checkbox"/> | 5. Palai <input type="checkbox"/> | |

84. YAKKAI

- | | | |
|---------------------------------------|--------------------------------------|--------------------------------------|
| 1. Vali <input type="checkbox"/> | 2. Azhal <input type="checkbox"/> | 3. Iyam <input type="checkbox"/> |
| 4. Valiazhal <input type="checkbox"/> | 5. Valaiyam <input type="checkbox"/> | 6. Azhalval <input type="checkbox"/> |

7. Azhaliyam ☐ 8. Iyavali ☐ 9. Iyaazhal ☐

10. Mukkutram ☐

85. GUNAM

1. Sathuva gunam ☐ 2. Rajo gunam ☐

3. Tamo gunam ☐

UYIR THATHUKKAL VALI

	1. Normal	2. Affected
86. Pranan	<input type="checkbox"/>	<input type="checkbox"/> _____
87. Abanan	<input type="checkbox"/>	<input type="checkbox"/> _____
88. Samanan	<input type="checkbox"/>	<input type="checkbox"/> _____
89. Udhanan	<input type="checkbox"/>	<input type="checkbox"/> _____
90. Viyanan	<input type="checkbox"/>	<input type="checkbox"/> _____
91. Nagan	<input type="checkbox"/>	<input type="checkbox"/> _____
92. Koorman	<input type="checkbox"/>	<input type="checkbox"/> _____
93. Kirukaran	<input type="checkbox"/>	<input type="checkbox"/> _____
94. Devathathan	<input type="checkbox"/>	<input type="checkbox"/> _____
95. Tananjeyan	<input type="checkbox"/>	<input type="checkbox"/> _____

AZHAL

	1. Normal	2. Affected
96. Anala pittham	<input type="checkbox"/>	<input type="checkbox"/> _____
97. Prasaka pittham	<input type="checkbox"/>	<input type="checkbox"/> _____
98. Ranjaka pittham	<input type="checkbox"/>	<input type="checkbox"/> _____
99. Aalosaka pittham	<input type="checkbox"/>	<input type="checkbox"/> _____
100. Saathaka pittham	<input type="checkbox"/>	<input type="checkbox"/> _____

IYAM

1. Normal

2. Affected

101. Avalambagam

☐☐

102. Kilethagam

☐☐

103. Pothagam

☐☐

104. Tharpagam

☐☐

105. Santhigam

☐☐**UDAL THATHUKKAL**

1. Normal

2. Affected

106. Saaram

☐☐

107. Chenneer

☐☐

108. Oon

☐☐

109. Kozhuppu

☐☐

110. Enbu

☐☐

111. Moolai

☐☐112. Sukkilam /
Suronitham☐☐**ENVAGAI THERVUKAL**

1. Normal

2. Affected

113. Naa

☐☐

114. Niram

☐☐

115. Mozhi

☐☐

116. Vizhi

☐☐Malam
117. Niram

1. Normal

2. Affected

☐☐

118. Thanmai

☐☐Moothiram
Neerkuri
119. Niram☐☐

120. Eadai ☐ ☐ _____

121. Manam ☐ ☐ _____

122. Nurai ☐ ☐ _____

123. Enjal ☐ ☐ _____

124. Neikuri: 1.Vatham ☐ 2. Pittham ☐ 3. Khabam ☐

4. Kalappu kuttram ☐ 5. Mukkuttram ☐

125. Sparisam: 1.Mithaveppam ☐ 2.Miguveppam ☐

3.Thatpam ☐

126. Naadi: 1.Vatham ☐ 2.Pittham ☐ 3.khabam ☐

4.Vathapittham ☐ 5. Vathakhabam ☐ 6. Pitthavatham ☐

7. Pitthakhabam ☐ 8. Khabavatham ☐ 9. Khabapittham ☐

10. Mukkuttram ☐

INVESTIGATION BLOOD

127. TC (cells /cumm):

DC (%): 128. P 129. L 130. E 131. B

132 M.

133. Hb (gms %):

ESR (mm/hr): 134. 1/2hr 135. 1hr

136. Blood Sugar (R) (mg %):

137. Blood Urea (mg %):

138. Serum Creatinine (mg %):

139. Serum Cholesterol (mg %):

URINE

140. Reaction: 1. Acid ☐ 2. Alkaline ☐

141. Albumin: 0. Nil ☐ 1. Trace ☐ 2. + ☐ 3. ++ ☐
4. +++ ☐

142. Sugar: 0. Nil ☐ 1. Trace ☐ 2. + ☐ 3. ++ ☐
4. +++ ☐

Deposit	1. Yes	2. No
143. Pus cells	<input type="checkbox"/>	<input type="checkbox"/>
144. Epithelial cells	<input type="checkbox"/>	<input type="checkbox"/>
145. RBC	<input type="checkbox"/>	<input type="checkbox"/>
146. Crystals	<input type="checkbox"/>	<input type="checkbox"/>

MOTION

	1. Yes	2.No
147. Ova	<input type="checkbox"/>	<input type="checkbox"/>
148. Cyst	<input type="checkbox"/>	<input type="checkbox"/>
149. Occult blood	<input type="checkbox"/>	<input type="checkbox"/>

150. **ULTRA SONOGRAPHY:**

151. ADMITTED TO TRIAL: 1.Yes ☐ 2. No ☐
If yes

152. S. No:

153. I.P / O.P 1. I.P ☐ 2. O.P ☐

154. Drug issued for OP patient (g):

155. Station

57. Signature of Medical Officer

156. Date:

NATIONAL INSTITUTE OF SIDDHA, CHENNAI – 47

AN OPEN TRIAL OF *KALLADAIPPU THOOL* FOR THE TREATMENT OF *KALLADAIPPU* (URINARY CALCULUS)

FORM II- ASSESSMENT PROFORMA

1. O.P.No / I.P No: _____ 2. Bed No: _____ 3. S.No:

--	--
4. Name: _____
5. Date of admission to the trial:

--	--	--	--	--	--
6. Date of assessment:

--	--	--	--	--	--
7. Week of assessment:

--

CLINICAL ASSESSMENT

SIGNS AND SYMPTOMS

8. Abdominal pain 1. No ☐ 2. Yes ☐
9. Severity: 0. NA ☐ 1. Mild ☐ 2. Moderate ☐ 3. Severe ☐
10. Sites: 0. NA ☐ 1. Loin ☐ 2. Renal angle ☐ 3. Lower abdomen ☐
11. Radiating areas: 0. NA ☐ 1. Groin ☐ 2. Thigh ☐
3. External Genitalia ☐

	1. Yes	2.No
12. Abdominal distention	<input type="checkbox"/>	<input type="checkbox"/>
13. Dysuria	<input type="checkbox"/>	<input type="checkbox"/>
14. Oliguria	<input type="checkbox"/>	<input type="checkbox"/>
15. Yellowish urination	<input type="checkbox"/>	<input type="checkbox"/>
16. Haematuria	<input type="checkbox"/>	<input type="checkbox"/>
17. Pyuria	<input type="checkbox"/>	<input type="checkbox"/>
18. Nausea	<input type="checkbox"/>	<input type="checkbox"/>
19. Vomiting	<input type="checkbox"/>	<input type="checkbox"/>
20. Fever	<input type="checkbox"/>	<input type="checkbox"/>
21. Sweating	<input type="checkbox"/>	<input type="checkbox"/>

INVESTIGATIONS (only at the end of treatment)

BLOOD

22. TC (cells /cumm):

DC(%): 23. P 24. L 25. E 26. B 27. M

28. Hb (gms %): .

ESR (mm/hr): 29. 1/2hr 30. 1hr

31. Blood Sugar (R) (mg %):

32. Blood Urea (mg %):

33. Serum Creatinine (mg %): .

34. Serum Cholesterol (mg %):

--	--	--

URINE

35. Reaction: 1. Acid ☐ 2. Alkaline ☐

36. Albumin: 0. Nil ☐ 1. Trace ☐ 2. + ☐ 3. ++ ☐ 4. +++ ☐

37. Sugar: 0. Nil ☐ 1. Trace ☐ 2. + ☐ 3. ++ ☐ 4. +++ ☐

Deposit

1. Yes 2. No

38. Pus cells ☐ ☐

39. Epithelial cells ☐ ☐

40. RBC ☐ ☐

41. Crystals ☐ ☐

MOTION

1. Yes 2.No

48. Ova ☐ ☐

49. Cyst ☐ ☐

50. Occult blood ☐ ☐

51. **ULTRA SONOGRAPHY:**

INVESTIGATION IN SIDDHA SYSTEM

NEERKURI

1. Normal

2. Affected

42. Niram

☐
☐

43. Edai

☐
☐

1. No

2. Yes

44. Manam

☐
☐

45. Nurai

☐
☐

46. Enjal

☐
☐

47. Neikuri: 1. Vatham ☐

2. Pittham ☐

3. Khabam ☐

4. Kalappu kuttram ☐

5. Mukkuttram ☐

52. Drugs returned (capsules):

--	--

53. Drugs issued (capsules):

--	--

54. Station: _____

56. Signature of the Medical Officer

55. Date: _____

CONSENT FORM

Certificate by Investigator

I certify that I have disclosed all details about the study in the terms really understood by the patient.

Date: _____

Signature: _____

Station: _____

Name: _____

Consent by Patient

I have been informed to my satisfaction, by the attending physician, the purpose of the clinical trial, and the nature of the drug treatment and follow-up including the laboratory investigations to be performed to monitor and safeguard my body functions.

I am aware of my right to opt out of the trial at any time during the course of the trial without having to give the reasons for doing so.

I, exercising my free power of choice, hereby give my consent to be included as a subject in the clinical trial of ***Kalladaippu Thool*** for *Kalladaippu*.

Date: _____

Signature: _____

Station: _____

Name: _____

Signature of witness: _____

Date: _____

Name: _____

Station: _____

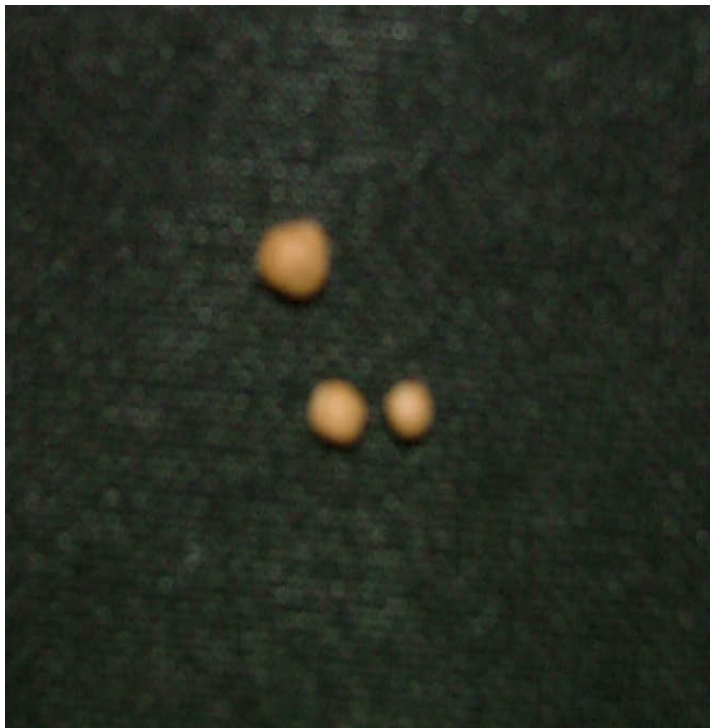
Relationship: _____

URINARY CALCULUS REMOVED IN URINE

O. P NO: U5278, Name: Mr. Raja



O. P. No: T101, Name: Mrs. Pattammal



O. P. No: S. 359, Name: Mr. Ranganathan



O. P. No: S. 511, Name: Mr. Anandan



ACHYRANTHUS ASPERA



ASH POWER OF ACHYRANTHUS ASPERA



MUSA PARADISIACA



ASH POWDER OF MUSA PARADISIACA



BORASSES FLABELLIFER



ASH POWDER OF BORASSES FLABELLIFER



SOLANUM MELONGENA



ASH POWDER OF SOLANUM MELONGENA



UNCARIA CAMBIR



STEM



POWDER FORM OF UNCARIA CAMBIR





Sodium baborate (Before purification)



Sodium baborate (Purified)

ALUMEN



PURIFIED ALUMEN



SODIUM CHLORIDE IMPURA



PURIFIED SODIUM CHLORIDE IMPURA



PURIFIED SOWKARAM



KALLADAIPPU THOOL



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8. Theriar gunavagadam
9. Anupogavaithiya navaneetham
10. Matchamuni 800
11. Gunapadam Mooligai
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12. Gunapadam thathu
-R. Thiyaragan
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